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SYSTEM

OF

DISEASES OF THE EYE.

BY

AMERICAN, BRITISH, DUTCH, FRENCH,
GERMAN, AND SPANISH AUTHORS.

EDITED BY

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OF PHILADELPHIA, PA., U.S.A.

VOLUME III.

LOCAL DISEASES, GLAUCOMA, WOUNDS AND INJURIES,
OPERATIONS.

WITH FIFTY FULL-PAGE PLATES AND ONE HUNDRED AND EIGHTY-SIX TEXT ILLUSTRATIONS.

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SYSTEM

OF

DISEASES OF THE EYE.

PART III.

LOCAL DISEASES, GLAUCOMA, WOUNDS AND INJURIES, OPERATIONS.

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DISEASES OF THE ORBIT.

BY CHARLES STEDMAN BULL, A.M., M.D.,

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ANOMALIES OF THE ORBIT.

CONGENITAL ANOMALIES OR MALFORMATIONS.

Malformations of the orbit are usually congenital, and form a part of the malformations of the skull or the malformations and arrests of development of the brain and eyeball. These malformations of the bony walls are in the form either of displacements or of arrest of development. In acephalous monsters the plane of the anterior orbital opening is deflected strongly upward, and the superior orbital margin is on a plane posterior to that of the inferior orbital margin. The lesser wings of the sphenoid which surround the optic foramen are very small, and are sometimes connected with the body of the sphenoid merely by periosteum. The orbital plate of the frontal bone is very defective, and the cavity of the orbit is directly connected with the cranial cavity and the pterygo-palatine fossa by a large, irregular opening which is formed by the superior and inferior orbital fissures.

In the double-faced monsters with three or four eyes the bony walls of the orbit are either in a condition of arrested development or are entirely lacking. In the form of monster known as hemicephalus with marked prognathism, the orbits are enormously enlarged. The horizontal portion of the frontal bone is very short, the ethmoid bone is narrowed and cartilaginous, and the lesser wings of the sphenoid are rudimentary and directed forward.

In cases of cyclopia, with a single common orbital cavity, the latter is rhomboidal in shape and usually of small diameter. The roof of the orbit is here formed by the frontal bone, the apex by the lesser wings of the sphenoid bone, the sides are made by the greater wings of the sphenoid and the zygoma, and the floor by the superior maxilla, which is very defective. The optic foramina are sometimes entirely wanting.

In cases of microphthalmos and anophthalmos the orbit is excessively narrowed and pushed to one side, the roof is defective, and the optic foramina are usually absent.

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After atrophy or loss of the eyeball in infancy or early childhood, the facial opening of the orbit becomes smaller in vertical diameter, and assumes more the shape of a fissure with its width elongated. The roof of the orbit becomes flattened, while the floor becomes somewhat convex, and the cavity is narrowed in all its diameters.

Among the defects of growth and development may be mentioned *congenital fissures, intercalary bones,* and the so-called *cribra orbitalia.*

The congenital fissures are found in those parts of the orbital walls which are developed directly from ossification of the cartilaginous nasal capsule,—that is, the *lamina papyracea* of the ethmoid bone and the *superficies orbitalis* of the superior maxilla.

Intercalary bones are met with in the roof of the orbit.

The cribra orbitalia are always bilateral, and are met with in the roof of the orbit, generally in children. They resemble osteophytes, but they are not pathological. Another very rare anomaly is ossification of the trochlea and displacement of the entrance to the orbit by a bony arch beneath the supra-orbital margin.

ACQUIRED ANOMALIES.

Acquired anomalies or malformations of the orbital walls are by no means rare as a result of disease. The form of the orbit may be materially altered by distention of some of the neighboring cavities. Of these the most frequently met with are hydrops and empyema of the frontal sinus, and these conditions are usually accompanied by ectasia of the ethmoid cells. Exophthalmos may be caused by this protrusion of the roof and inner wall of the orbit inward, though the motility of the eyeball is usually unimpaired. Similar distention of the sphenoidal and maxillary sinuses rarely produces any effect upon the shape of the orbit.

The complete absence, the arrest of development, or the excessive development of one or more of the bones forming the cavity of the orbit may cause great differences in the general shape of the cavity. If there is a partial or total arrest of development in the orbital portions of the frontal, superior maxillary, ethmoid, nasal, or lacrymal bones, the two orbital cavities will bear a very varying relation to each other. The most marked anomaly of this kind is where the two orbits are fused into one, forming what is called cyclopia. When the ethmoid cells are directly continuous forward with similar cells in the frontal end of the ascending apophysis of the superior maxilla, the two orbits are abnormally separated from each other, and this abnormal distance is particularly marked when this ascending apophysis, together with the corresponding nasal bone, is flattened. This condition of the orbital walls is frequently accompanied by epicanthus. Sometimes all the bones which enter into the formation of the lateral walls of the orbital cavity are rudimentary on one side (monopsia) or both. Associated with this anomaly we sometimes meet with complete absence of the orbital cavities (so-called *imperforate orbit*).

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In hydrocephalus the bottom of the orbit is pushed forward by the intra-cranial fluid and causes exophthalmos.

We sometimes meet with an excessive protrusion forward of the orbital portion of the frontal bone. In both of these last conditions there is usually more or less continuous lacrymation, due to some displacement of the lacrymal puncta.

Meningocele is one of the anomalies of arrest of development of the orbit which has been occasionally met with. In this condition there is a defective development of the upper and inner angle of the anterior part of the bony wall of the orbit, consisting in an absence of part or all of the nasal process of the frontal bone and the adjacent part of the superior orbital ridge. As a consequence the meninges protrude and present as a tense, elastic, globular swelling, which beats synchronously with the heart, and is present from birth. This swelling can be reduced almost entirely by firm and steady pressure, and after its reduction the deficiency in the bony wall can be readily made out. The swelling might be mistaken for a dermoid tumor or a sebaceous cyst or a nævus. The absence of any bony defect after reduction of a nævus, or the displacement of the cyst, as well as the absence of pulsation, will prevent any error in diagnosis.

LESIONS OF THE BONY WALLS OF THE ORBIT.

The lesions which are met with in the bony walls of the orbit are: 1. Periostitis or osteo-periostitis, with or without subperiosteal abscess. 2. Caries and necrosis, involving more or less of the entire thickness of the bony walls. 3. Periostosis, hyperostosis, or exostosis of one or more bones. 4. Gumma or syphiloma of the periosteum.

PERIOSTITIS OR OSTEO-PERIOSTITIS.

Periostitis or osteo-periostitis is usually limited to the surface of bone, to the periosteum and superficial bone-layers. Beneath the periosteum, between it and the bone, are accumulated large numbers of round cells, analogous to the cells of the embryonic medulla. At the same time the deep layers of the periosteum are inflamed, and contain cells between the fibrous bundles. The neighboring connective tissue generally shows some inflammatory œdema, which accounts for the thickening and adhesions observed between the bone and the skin. When the bone-disease has lasted a long time, the round cells nearest the surface of the bone, beneath the periosteum, act like osteoblasts during the period of ossification, and in fact are practically identical with them. In other words, simple osteo-periostitis of the bones of the orbit consists in the return of the cells between the bone and the periosteum to the embryonic state. The varieties of new osseous products consequent on periostitis, such as osteophytes, exostoses, enostoses, and eburnation, constitute the accidents common to all forms of osteo-periostitis. The exuberant formation of the new osseous lamellæ

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under the periosteum may develop exostoses of varying size, and in the bone itself a parenchymatous hyperostosis and eburnation.

In syphilitic patients a gummatous osteo-periostitis or periosteal gumma is sometimes met with. This process is a variety of rarefying osteitis in which the subperiosteal embryonic tissue takes on the disposition observed in gummata. Gummata of bone are merely an intense and limited osteo-periostitis, with destruction of the osseous lamellæ by a rarefying osteitis. Though common in the other cranial bones, they are rare in the bones of the orbit. When a thin section of such a gumma is examined under a high-power objective, there is seen to be a tissue formed of very fine fibres, between which are found round cells with a nucleus and a small amount of protoplasm. These round cells, from 0.010 to 0.015 of a millimetre in diameter, are embryonic cells. Besides these cells, there are other cells of a fusiform or irregular shape, and still others, much smaller, apparently atrophied, measuring from 0.005 to 0.006 of a millimetre in diameter, almost entirely filled by their nuclei, and embedded in a finely granular matrix. What are called external gummata originate beneath the periosteum, and by pressure gradually detach it from the bone. They also press upon the underlying bone and enter its substance in the form of a cone, the bone becoming infiltrated and progressive rarefaction going on up to a certain varying point. Then the gumma ceases to advance, undergoes lardaceous or fatty metamorphosis, and finally disappears, leaving in its place a more or less extensive depression in the bone. Pathology teaches that the frontal bone is the region of predilection for these gummata.[1] When a small gumma of the orbital periosteum has formed a cavity in the bone, and the inflammatory process is stopped, the new material becomes caseous and atrophies, the peripheral osteo-periostitis heals, and there may be a partial reparation of bone-tissue. The cavity is not entirely filled, but the osteophytes developed beneath the periosteum end in a bony neoplasm at the margin of the loss of substance. If, however, the bone is completely perforated, the defect is not filled up by new bone-tissue, but by a fibrous cicatrix.

The two essential signs of syphilitic orbital osteo-periostitis are pain and swelling, the former most intense at night and sometimes very violent. To these two symptoms is added a third, exophthalmos, if the bony lesion be extensive or situated deeply in the orbit. Neuralgia, due to compression of a nerve-filament at some point in its passage through a bony canal, is a very common symptom.

Osteo-periostitis of the bones of the skull produces a tumor generally broad and flattened. Subperiosteal gummata, extensive and thick, are generally accompanied by inflammation of the skin, long suppuration, and necrosis of portions of bone which remain imprisoned for a long time, and which are eventually cast off, leaving behind great losses of substance. The

[1] See a monograph by Moscovits, De la Syphilis Tertiaire Crânienne, Paris, 1874.

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diagnosis of these gummata is not always easy. If the skin is adherent at a point to the bone, periostitis is almost certainly present, and if a tumor is felt, it is certainly a gumma or a periostitis, according as the consistence of the tumor is elastic or bony. Hueter holds that the formation of a sequestrum rarely occurs here, owing to the slight tendency of the periosteum in the orbital region to the new formation of bone. The caries in these cases is, however, generally due to the suppuration of syphilomata. The discolored and dead bone does not fall off as a sequestrum, but small granulations come from the diploë of the bone, perforate this dead cortical layer, and push it aside, and it subsequently necroses in small particles. The syphilitic tissue proliferation does not, however, always end in suppuration, but sometimes forms a sclerosed bony substance, to which the name of syphilitic osteoma has been given. These osteomata are generally smooth, flat, and limited in extent.

Though these various lesions of the orbital walls are generally regarded as late manifestations of syphilis, they still sometimes occur as early lesions, and this is particularly true of periostitis. (See Mauriac and Fournier.) Orbital periostitis may be one of the first signs of constitutional infection, occurring sometimes shortly after the appearance of the initial lesion. It is confined almost exclusively to the periosteum, and whatever inflammatory condition of the bone may be present is merely secondary to the periostitis. In the adult these periosteal swellings tend to spontaneous resolution, and they often rapidly disappear without leaving any trace. In the hereditary syphilis of children, however, this form of periostitis very soon takes on suppurative action, and ends in the death of the bone. In acquired syphilis this early form of orbital periostitis is circumscribed, varies in duration between four and six weeks when left to itself, and disappears much sooner under appropriate treatment. These precocious bone lesions of the orbit are much more common and severe in hereditary syphilis. They bear no resemblance to the later periostitis, being much less indolent, lasting a shorter time, tending to spontaneous resolution, and but little to destructive metamorphosis. They are never followed by hyperostosis or exostosis, and never leave any trace of their presence. Large, hard frontal bosses, which may involve the entire supra-orbital region and dip down deep into the orbit, rapidly disappear under appropriate treatment.

Symptomatology.—Two forms of inflammation, acute and chronic, must be distinguished. The acute form is almost always a precocious lesion, and the symptoms are apt to be severe. The patient complains of great pain in and around the eye, especially along the superior orbital margin, which is excessively sensitive to pressure, even when the periostitis is deeply seated and does not involve the orbital margin. The eyelids are red and swollen, and the ocular conjunctiva is injected and sometimes chemotic, the latter symptom being much more marked if the cellular tissue of the orbit is involved. There is more or less protrusion of the eyeball, according as the seat of the periostitis is deep in the orbit or near its margin; but the

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exophthalmos is rarely straight forward, owing to the periostitis being confined to one wall or portion of the orbit. The general constitutional signs of inflammation are usually severe, as the cellular tissue of the orbit becomes involved. There may be partial or complete loss of vision from pressure on the optic nerve in the optic foramen or in the orbit, or loss of the eye by strangulation of the entire blood-supply of the eyeball by the infiltrated orbital tissue. Optic neuritis followed by atrophy of the nerve, extending to the sheath of the nerve from the periosteum of the orbit, and simple atrophy of the optic nerve from pressure by the surrounding infiltrated tissues, are not very uncommon results. In such cases the ophthalmoscope would show either the signs of papillitis or of inflammatory atrophy of the disk, or the signs of simple gray discoloration of the disk, without the appearances of papillitis. When the orbital cellular tissue becomes inflamed, there is much more probability of the formation of pus than when the periosteum is alone involved. The attack may be ushered in by a chill, followed by high fever, and all the other symptoms are then rapidly developed. The pain may, however, occur only periodically at certain times of the day.

In the *chronic* form the inflammatory symptoms are far less pronounced, and the disease is more protracted and insidious in its course. The lesion in the bone is often developed very slowly, and when deep-seated is often overlooked. There may be little or no febrile excitement, but slight pain, no protrusion of the eyeball until late in the course of the disease, and in fact scarcely any objective symptom unless the periostitis is near the orbital margin. Here the œdematous swelling of the surrounding parts, the decided thickening of the bone, and the presence of a hard, indistinctly fluctuating tumor which is painful and sensitive, all aid in the diagnosis. The chronic form is very often accompanied by the formation of a subperiosteal abscess, which sometimes strips up the periosteum from the bone for a long distance, and is likely to end in caries and necrosis of the bone. The abscess thus developed tends to open at a point outward through the conjunctiva or lid by one or more sinuses. If the purulent process has been so extensive that caries has occurred in several directions, the abscess may open into the nose, the frontal sinus, the maxillary sinus, or the cavity of the skull. If the abscess perforates through the conjunctiva, it is almost certain that the seat of the bone lesion is deep in the orbit; for if the margin of the bony orbit were involved, the opening would be in front of the tarso-orbital fascia, through the lid. If the tendency to suppuration be but slight, the periosteum may become very much thickened, and small nodules or periosteal growths may be developed, which may ossify and form true exostoses. These may exist between the periosteum and the bone, or more rarely they may grow in both directions, outward towards the orbit and inward towards the bone. One dangerous symptom which might seem to be imminent very rarely occurs,—that is, an extension of the periosteal inflammation to the meninges of the brain through the medium of the optic

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foramen. It is far more likely to induce meningeal inflammation when the roof of the orbit is the seat of the lesion, for here the bone is quite thin, and a carious process once started in this region may soon perforate the bone and open into the anterior fossa of the skull, leading to meningitis or abscess of the brain.

Where a sinus exists, the introduction of a probe proves the roughness of the bone, and this, with the continued patency of the sinus, proves that the disease has passed from periosteum to bone. Pieces of loose bone are but rarely found; for though the bone is very thin, it is hard, and we are more likely to meet with a perforation communicating with some neighboring cavity. Still, this complication is uncommon, for the caries is almost always superficial, and the case ends in recovery after the carious bone has been cast off or has lost its roughness and the sinus closes.

Treatment.—In acute cases hot fomentations and perhaps poultices must be diligently applied, and internally quinine and iron must be administered, and, if the patient is a child, cod-liver oil may be added. If the case be of syphilitic origin, potassium iodide in large doses, twenty to thirty grains three times a day, is indicated. If rheumatism be the probable cause, sodium salicylate or potassium iodide in small doses should be prescribed. If suppuration threaten, or an abscess actually form, a deep early incision should be made with a long straight bistoury, and the abscess gently irrigated with a warm antiseptic solution, and then the incision kept open by a pledget of cotton or a drainage-tube. It is very necessary that the external opening of the incision be large, and this must be kept freely dilated. The irrigation should be done several times a day. If rough or dead bone is discovered by the probe, its removal must not be attempted, at least until all signs of acute inflammation have subsided and a large, free opening exists down to the bone; and this rule is especially applicable when the diseased bone is in the roof of the orbit.

In *chronic* periostitis the treatment must be modified in accordance with the severity of the symptoms. Hot compresses may be used as a soothing application, but their application need not be constant, and our main reliance should be placed on the internal treatment by sodium salicylate or potassium iodide.

CARIES AND NECROSIS.

Caries and necrosis of the bones of the orbit occur most frequently at or near the orbital margin, or at the upper and inner corner of the roof. While most of these cases begin with a periostitis or an osteo-periostitis, the lesion may develop in some cases as a real osteitis and involve the periosteum secondarily. These are the worst cases, for the disease is usually situated deep in the orbit, and has probably spread to the bones of the orbit from some other part of the skull. It is then very apt to involve the orbital cellular tissue and end in prolonged suppuration. The lesion has been known to spread from a syphilitic ozæna in the nose, extending to the

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ethmoid cells, thence to the orbital plate of the ethmoid, and finally to the orbital cellular tissue. The process is here very chronic, even under the most rational and persistent treatment, and results in extensive exfoliation of bone and considerable deformity. In these severe cases several sinuses may form in the lids or surrounding structures in different directions, through which pus and small fragments of exfoliated bone may be discharged for an indefinite period, ending either in deeply retracted cicatrices or in eversion of the eyelids. In some cases the adhesions between the bone, periosteum, and external soft parts, as the eyelid, have been found so dense and firm that any operative attempt to separate them has proved to be impossible. One of the commonest causes of this extensive caries of the orbital walls is constitutional syphilis. Though a late manifestation of the disease, it has been known to occur among the precocious lesions. It is always a chronic process, and a rapid cure cannot be expected.

Treatment.—Just how far it is justifiable to operate for the removal of carious bone in the orbit is a somewhat difficult matter to determine. Cases have occurred in which the carious process has involved the ethmoid bone and the roof of the orbit and has opened into the ethmoid cells and the anterior fossa of the skull. In the latter case the condition of the patient is desperate, for the purulent process may extend directly to the meninges and kill the patient, or some loosened fragments of bone may set up meningitis by mechanical irritation, or the carious process in the bone may set up a subdural or cerebral abscess, even without perforation of the roof of the orbit, through the media of the fine foramina for the passage of the nutrient blood-vessels. The safer and better plan is to remove all pieces of loosened bone, even from the roof of the orbit, through a free opening, thus doing away with one source of cerebral irritation, and bringing about free drainage. If the ethmoid be involved, it is best to remove as much of the diseased bone as can be reached, making a free opening into the ethmoid cells. This removes one source of continuous irritation and renders easier the introduction of a drainage-tube through the nose, which is a very necessary procedure. The same thing holds true of the lacrymal bone, which, however, is not so frequently the seat of carious disease as the ethmoid, unless from chronic dacryocystitis and disease of the nasal duct. After the removal of all pieces of loose bone, and the establishment of free drainage through the nose by breaking down the bony structures and enlarging the openings leading to the superior nasal meatus, the whole cavity should be carefully and repeatedly washed out by a solution of mercuric bichloride (1 to 2000), and then a drainage-tube should be inserted from above, and if possible brought out through the nose. This should be made of rubber and fenestrated, and left in place as long as proves necessary.

PERIOSTOSIS, HYPEROSTOSIS, AND EXOSTOSIS.

According to modern authorities, hyperostosis and periostosis are two distinct pathological processes in bone, though they may, and very often do,

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coexist, especially in the bones of the face and skull. Hyperostosis is rare in the region of the orbit, for out of a total of nearly twenty thousand cases of eye-disease seen by the writer, it has been observed in only four cases. The excessive development of one or more of the bones which enter into the formation of the orbit must produce the most singular changes in the shape of this cavity.

Hyperostosis is an increase in the diameter of the bone, occurring in the whole bone or in a part of it. It may affect only the external compact tissue, or the medullary substance alone, or may be met with in both at the same time. These processes occur more frequently in the skull than anywhere else in the body. The hyperostosis of the cranial bones seems to involve mainly the external surface of the bones. It was formerly supposed that some inflammatory symptoms must accompany or precede this process, but it is now known that the thickening of bones may go on for an indefinite period after the cessation of all symptoms of inflammation. Some authors make a distinction between hyperostosis and simple hypertrophy, the latter being defined as an increased development of the characteristic elements of bone-tissue, while the former is described as an altered relation of these elements to one another. As a result of the change in size and shape of the bones of one orbit, the two orbits do not occupy a corresponding position, but one of them is found to be on a higher plane than the other. This difference may be very great and yet the functions of the two eyes may be perfectly carried out. It has been supposed that this abnormal growth is due to some perverted nerve-action which influences the nutritive changes going on in the parts, but this theory cannot apply to all cases, for the abnormal growth would be confined to the periosteum, which is not always true. Instances of periostosis joined to hyperostosis, as a result of chronic traumatic periostitis, are more common than those of pure hyperostosis.

Periostosis signifies a thickening of the periosteum, existing alone without any hyperostosis, and probably occurs in the following way. A periostitis may have been set up by some traumatism, and the underlying bone after a time becomes inflamed. If this inflammatory process in the bone be arrested before caries sets in, the consequence will probably be a hyperostosis. To this process the bones of the orbit are said to be peculiarly liable, though clinical observation by no means confirms this statement.

Perhaps the most interesting cases of orbital disease due to syphilis are those which present the results of chronic hyperplastic bone-disease, on account of their rarity and of the possible resulting deformity. Periostosis is a rare process in the orbit, where periostitis usually either yields to treatment and leaves no trace of its presence, or else ends in suppuration and caries. Periostosis here probably begins as a chronic periostitis, which ends in induration or sclerosis, forming a more or less circumscribed tumor along the orbital margin, and but very rarely in the deeper parts of the orbital cavity. The syphilographers distinguish three kinds of periostosis,—inflammatory, plastic, and gummatous; but the second variety is merely a

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stage of the first, while the third is probably a periostitis with the formation of a subperiosteal gumma. As regards the inflammatory periostosis, the term *node* would apply as well to circumscribed periostoses of the orbit as it does to periostoses in other parts of the body. Such periostoses are usually late manifestations of syphilis, the result of long-continued plastic inflammation of the periosteum, and only in isolated instances ending in ossification. If they happen to occur in the vicinity of the supra-orbital or infra-orbital foramina, there is more or less constant neuralgia in the region of distribution of the nerves passing out through these foramina, which increases in severity as the periostosis extends. Though they are almost always observed along the orbital margin, they may occur deep in the orbit, at or near the apex and around the optic foramen. It is probable that some of the cases of paralysis, partial or complete, of one or more of the ocular muscles, coming on somewhat gradually, are due to a periosteal node pressing on the muscle or its nerve-twig in its course or near its origin, producing paralysis by direct pressure as it grows. Such a node growing from the periosteum at the extreme apex of the orbit, if of any size, might easily involve the origins of all the straight muscles of the eye, without any very great projection into the cavity of the orbit. In such a case the optic nerve would, of course, be involved, and there would be atrophy of the nerve, perhaps preceded by neuritis. The occurrence of such cases would offer a plausible explanation of the reason why so many cases of paralysis of the ocular muscles in syphilitic patients are not cured by appropriate anti-syphilitic treatment. The periosteal thickening goes on gradually, involving the origin of the muscle or its motor-nerve branch, until the latter becomes atrophied from compression, and then the mischief has been done and the paralysis is permanent, although in favorable cases the periostosis may be absorbed by treatment.

Another symptom which may be produced by periostosis deep in the orbit is exophthalmos.

This form of periostitis involved in periostosis does not tend to spread, and hence is but little likely to involve the orbital tissue. Any protrusion of the eyeball is here due to the periostosis itself. There is no sign of acute inflammation, no constant pain in the orbit, and no sensitiveness to pressure along the orbital margin. On pressing the eye backward pain is experienced, but the process may continue from the beginning without any pain, and the patient's attention may be first attracted by the exophthalmos, more or less limitation in motility of the eye, diplopia, and, finally, impairment of vision.

Hyperostosis is the rarest of all the affections of the bones of the orbit. It differs from periostosis in being primarily a disease of the bone itself, involving the periosteum only secondarily. It may affect the entire thickness of the bone, and may produce very singular changes in the shape of the orbit. It is a hyperplasia of bone, distinct from exostosis, is of ivory hardness, and never yields to any constitutional treatment. Syphilis is not

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a common cause of its development. Any operation for the removal of the enlarged bone or bones is justifiable only when its mechanical presence as a hinderance to the functions of the eye demands it. Its removal is then best effected by means of a mechanical drill, such as is employed by dentists.

Exostosis.—Orbital exostoses are outgrowths from the periosteum or bones of the orbital walls towards the orbital cavity. They differ from the swelling and projection of periostosis both in shape and in size, and somewhat also in location. Though occurring in all parts of the orbit, they are more frequently met with on the inner wall and near the margin of the orbit than elsewhere. An exostosis is usually smaller than a periostosis, with a narrower base, but it projects farther into the cavity of the orbit. These exostoses are always covered by periosteum, and frequently do not involve the subjacent bone at all. They occur more frequently than periostoses or a hyperostosis. They may develop in consequence of long-continued chronic periostitis, and may exist simultaneously with a periostosis, though the latter is almost always the earlier in appearance of the two. They may, however, occur alone as direct outgrowths from beneath the periosteum, with or without signs of circumscribed inflammation. If they are situated deeply in the orbit, the most marked symptom is exophthalmos. The os planum of the ethmoid is a favorite seat of exostoses. When they occur anteriorly and admit of digital examination, they are recognized as hard, smooth elevations, with circumscribed base, not usually painful on pressure, but generally causing pain by pressure on the eyeball or upon the nerves within the orbit. Though usually slow in growth, and very often a late manifestation of syphilis, they may advance rapidly in size and be accompanied by some of the signs of acute localized inflammation. Several of these small exostoses have been known to appear on the inner wall or floor of the orbit, and, after pursuing a short, isolated existence, have coalesced to form one large projection, which interfered seriously with the movements of the eyeball. The more deeply in the orbit these exostoses are situated the more likely are they to escape attention, unless of such a size as to cause exophthalmos. When near the apex of the orbit, their pressure on the ciliary nerves or the ophthalmic branch of the trifacial causes deep-seated pain, which may be located in the cavity of the skull, and excite suspicions of the presence of an intra-cranial tumor, or perhaps meningitis. If the pressure is so extensive as to interfere with the return circulation from the eyeball, the ophthalmoscope may reveal the presence of papillitis or atrophy of the optic disk, both of which conditions might go far towards strengthening the suspicion of the existence of serious intra-cranial trouble. The history of the case, and the favorable results of a rigid antisyphilitic treatment in the subsidence of the symptoms, would perhaps aid in locating the disease within the orbit, although all these symptoms, with the exception of the exophthalmos, might be caused by an exostosis at the base of the skull.

Inasmuch as the usual cause of orbital exostoses is constitutional syphilis, the treatment should at first always be constitutional. Although less favor-

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able results are gained from the medical treatment of exostoses than of periostitis, they still sometimes disappear from the orbit very rapidly under the combined use of mercury and potassium iodide. If it becomes advisable or necessary to resort to operative interference for the removal of these exostoses, the soft parts should first be carefully dissected away from the bony projections. Then a pair of strong bone-forceps will usually suffice to remove them, as the base of attachment is generally slender. If the latter be stout and broad, it may become necessary to employ the chisel and mallet.

INJURIES OF THE ORBIT.

INJURIES OF THE ORBITAL MARGIN.

Contusions of the margin of the orbit are frequently followed by more or less extensive extravasations of blood, which may be either subcutaneous, subaponeurotic, or subperiosteal. They may be confined entirely to the point of impact or may extend in every direction under the skin, and in severe cases even under the conjunctiva. If an artery of any size has been injured, the extravasated blood forms a pulsating tumor which may render the diagnosis extremely difficult. Incised and lacerated wounds of the soft parts overlying the bony margin of the orbit are very common accidents, and, if seen soon after their occurrence, heal readily under antiseptic treatment, in spite of the crushing of the tissues, and by the aid of a few sutures generally heal by first intention.

Blows upon the margin of the orbit frequently involve the supra-orbital and infra-orbital nerves, and to the laceration of one or both of these nerves has been attributed the amaurosis, or loss of sight, which has been known to follow such injuries. Amaurosis from paralysis and subsequent atrophy of the optic nerve, following injuries of these branches of the trifacial, has long been recognized in ophthalmology; but the doctrine of the hypothetical connection between laceration of some fibres of the fifth nerve and paralysis of the optic nerve rests upon very insufficient evidence, and is now not even regarded as plausible. The real cause, at least in the great majority of the cases, is probably a fissure or fracture of the orbit extending backward to the optic foramen. Injuries of the bony margin of the orbit are generally connected with direct or indirect fractures of the orbital walls or bones of the skull, though instances are on record of small portions of the bony margin having been chipped off by blows, without the extension of the fracture backward. These cases are comparatively trivial in their nature and do well.

Fracture or dislocation of the malar bone may occur from a violent fall upon the malar prominence, or from a blow upon the same region. Such an accident usually fractures the zygoma, and sometimes opens the suture between the malar and superior maxillary bones, and may cause extensive hemorrhage into the orbit. Anæsthesia of the infra-orbital nerve,

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from laceration of the nerve in its bony canal, and annoying diplopia, from injury to the inferior oblique muscle, are occasional consequences of fracture of the malar bone.

FRACTURE OF THE ORBITAL WALLS.

Fracture of the bones of the orbit may be either direct or indirect, and may be caused by blows or falls upon the head or face, gunshot wounds of the orbit, and penetrating wounds of the orbit, with or without the lodgement of a foreign body. It is not always possible, even in direct fractures, to determine whether the injury is simply a fissure through the bone or a fracture with displacement of the fragments, though we may infer the latter from some of the accompanying symptoms. The usual symptoms are hemorrhage and displacement of the eyeball. If the hemorrhage is extensive, causing exophthalmos and ecchymosis of the lids and conjunctiva, there is certainly a fracture, with displacement of the fragments. Fractures of the outer wall are generally more easily recognized than those elsewhere by the probe and finger and by crepitation which can be induced in this region, as well as by laceration of the overlying soft parts. Indirect fractures rarely occur in the outer wall alone, but are usually accompanied by fracture of the inferior orbital margin. Direct fractures of the outer wall are usually produced by gunshot wounds.

Gunshot wounds of the orbit greatly complicate the diagnosis of the lesions produced, because of the extension of the injury to the other walls of the orbit and its contents, especially the eyeball and the optic nerve. Where sudden and immediate blindness occurs after a gunshot wound of the orbit, without any apparent injury to the eyeball, it may be due to laceration or complete rupture of the optic nerve by the bullet or by displaced fragments of bone, or to rupture of the choroid through the region of the macula, the other coats of the eyeball being intact.

Fractures of the *inner* wall of the orbit may occur alone or in connection with fracture of the other walls. They may be direct or indirect, and may involve one or both orbits. In the latter case the fracture of the inner wall is complicated by fracture of the horizontal plate of the ethmoid and of the roof of one or both orbits. The diagnosis of direct fracture is easily made by the displacement of the lacrymal bone from the orbital plate of the ethmoid. If this cannot be made out, bleeding from the nose and emphysema of the tissue of the orbit and eyelids will assist in the diagnosis. The latter is an important and valuable symptom.

Fracture of the *floor* of the orbit rarely occurs alone, but is usually connected with fracture of the malar and superior maxillary bones. The symptoms are hemorrhage into the orbit, bleeding from the nose or mouth (the blood coming from the maxillary antrum), emphysema of the orbit, and displacement of the eyeball by the broken fragments.

One of the almost constant results of fracture of the superior maxilla

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is anæsthesia of the infra-orbital nerve, which is usually permanent and due to complete rupture of the nerve.

Fractures of the *roof* of the orbit may be both direct and indirect, and are the most serious of all injuries to the orbit, on account of the possible consequences. These fractures may involve the orbital margin, or the lesion in the bone may be far removed from the margin, as in the cases caused by penetrating wounds with a cane or knife. The latter are the most dangerous. Berlin reports fifty-two cases of such perforation of the roof of the orbit, of which forty-one proved fatal. The objective symptoms are here usually wanting, unless the superior orbital margin is involved in the line of fracture. If the brain has been directly lacerated, brain-substance may protrude from the wound. When cerebral symptoms occur, they may be due either to pressure or to inflammation, and there will be pain, vertigo, delirium, paralysis, and coma. Sometimes the head-symptoms may be so long delayed that the surgeon begins to doubt the existence of perforation of the roof of the orbit, and yet in the end they appear, and the case is almost certain to end fatally. In the forty-one fatal cases reported by Berlin, the autopsy showed that the perforation in the roof of the orbit was small, and the bony fragments were in the cranial cavity. Though the perforation may be small, there may be very extensive laceration of the brain-tissue. Death may result from meningitis, or abscess of the brain, or thrombosis of the longitudinal sinus. All varieties of fracture of the roof of the orbit are marked by more or less prolonged signs of concussion of the brain and hemorrhage into the orbital tissue.

Leaving out of consideration those cases of fracture of the walls of the orbit which terminate fatally soon after receipt of the injury, the *prognosis* of cases of injury to the orbital walls with reference to vision is always very uncertain. Such cases may properly be divided into three classes: 1. Those in which impairment or loss of vision occurs immediately on receipt of the injury and remains permanent. 2. Those in which the impairment of vision occurs at once, but improves as time elapses. 3. Those in which the impairment of vision subsequently makes its appearance. In the first class of cases the injury has almost always been a fall or severe blow directly upon the skull, usually on the frontal bone. The first symptom which the patient notices on regaining consciousness is blindness in the eye on the corresponding side, and this sudden blindness is usually complete and permanent. This may not be accompanied by any ophthalmoscopic change, but eventually there are all the signs of atrophy of the optic nerve, either simple or inflammatory. The cause of the blindness is here usually fracture of the roof of the orbit and optic foramen, connected with fracture at the base of the skull. Von Hölder has published the results of his examination of the bones in one hundred and twenty-four cases of fracture of the skull. Among these there were eighty-six of fractures of the base, and in seventy-nine of these the fracture involved

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the roof of the orbit. In sixty-three cases there was a fissure or fracture running through the optic foramen, always through its upper wall, sometimes also through the inner wall. In forty-two cases there was hemorrhage into the sheath of the nerve, and he never found blood in the optic sheath unless the bony canal was fractured. The hemorrhage was sometimes found in both optic sheaths.

When the blindness is unilateral, it always occurs in the eye corresponding to the injured side. It is due to laceration of the optic nerve by the fractured bones, or to atrophy produced by pressure on the nerve-fibres by the extravasated blood. The blood may come from the intra-cranial cavity, or from the vessels of the sheath of the nerve, or from laceration of the central retinal artery, and the extravasation is almost always found within the sheath. Callan has recently reported nine cases of injury to the orbital walls, resulting in immediate unilateral blindness, in which it was believed that the line of fracture ran through the optic foramen. He calls attention to the fact that the frontal bone unites with the nasal, superior maxillary, lacrymal, and ethmoid bones by a continuous line of sutures, until the lesser wing of the sphenoid is reached, and at this point the suture line bifurcates, forming an obtuse angle, and quite near the apex of this angle lies the optic foramen. The jar caused by a blow upon the orbit would find its weak point along this line of sutures, and the first point of resistance would be at the bifurcation adjacent to the optic foramen. The jar could not follow both lines with equal force at and beyond the bifurcation, and consequently the unequal strain would result in fracture of the bone in the optic foramen, causing compression or laceration of the optic nerve.

In those cases in which the fracture extends into the middle fossa of the skull, the blindness may be caused by direct laceration of the optic chiasm or optic tract, or by extensive extravasation of blood at the base of the skull, causing compression of the chiasm or tract. In such cases both eyes are affected.

If the fracture of the optic foramen has caused extensive hemorrhage into the sheath of the optic nerve, the primary ophthalmoscopic symptoms would be venous hyperæmia, arterial ischæmia, and fresh hemorrhages on the disk, in the retina, and perhaps also in the vitreous. The secondary ophthalmoscopic symptoms would be neuritis or neuro-retinitis, ending in atrophy of the papilla.

Treatment of Fracture of the Orbital Walls.—This is to be conducted on general surgical principles. Loose bits of bone which can be readily reached are to be removed, and the track of the wound is to be carefully irrigated with some antiseptic solution. If abscess of the orbit develop, it must be immediately laid open and free drainage maintained. If the roof of the orbit be perforated and symptoms of brain-trouble appear, further exploration of the wound may be indicated, and should be carried out under the strictest antiseptic precautions. Almost all the cases in this latter class terminate fatally.

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INJURIES OF THE SOFT PARTS OF THE ORBIT.

Under this head we include injuries not only of the orbital tissue, but also of the eyeball, muscles, optic nerve, and lacrymal gland. In many cases of injury to the orbital walls the contents of the orbit are also wounded. The eyeball is usually displaced and very often limited in its motility. Some of the fatty tissue of the orbit protrudes through the wound in the conjunctiva or cul-de-sac. Sometimes the lacrymal gland is lacerated or displaced and protrudes through the wound.

Foreign bodies, even of large size, may enter the orbit and pass entirely out of sight, and are often extremely difficult to find. They may lodge in the back part of the orbit, or far to one side, without destroying the eyeball, and, if they do not penetrate the cavity of the skull or some other of the neighboring cavities, may remain for a long period undiscovered. All such foreign bodies which can be seen or felt should be immediately removed and the wound treated antiseptically. But it is very often difficult to decide whether a foreign body has entered the orbit. When it is small, as in the case of shot or small bullets, grave doubt may exist as to its actual presence in the orbit, and this is especially true of bits of glass or gravel. The place of entrance is very small, closes instantly, and it is almost impossible to trace the foreign body. The wound is apt to be sinuous, and probing it is very unsatisfactory. If the case is seen within a short time after the receipt of the injury, careful probing of the wound will sometimes discover a small foreign body, and in recent injuries this should always be done. Its presence is sometimes indicated by a localized swelling, and if this subsequently develops into an abscess and spontaneously discharges, a fistulous opening remains which betrays the presence either of some foreign body or of dead bone. One of the most constant symptoms of the presence of a foreign body is displacement and limited motility of the eyeball. A later symptom is disturbance of vision caused by pressure on the eyeball or optic nerve, either directly or through the medium of a hemorrhage or inflammatory exudation. If the removal of a foreign body is followed by restoration of the vision, we may reasonably conclude that no permanent injury has been done. If the foreign body be sought for and found immovable, it has probably perforated one of the bony walls of the orbit, and great care must then be taken in its removal, for fear of causing further injury; this is especially important when the foreign body has been driven through the roof of the orbit. If the case is an old one, and the eyeball has become blind and phthisical, no attempt should be made to remove the foreign body unless the eye is first enucleated, as the foreign body is certainly encapsulated and practically innocuous. The toleration of the presence of foreign bodies of large size manifested by the orbit is sometimes extraordinary. Many such cases have been reported, and one of the most interesting has been published by Dr. H. D. Noyes in the *American Journal of the Medical Sciences* for July, 1882. A young man was injured by the

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explosion of his gun, and the breech-pin broke through the nose and disappeared. It was not known that any foreign body had entered the orbit, though some sinuses continued to discharge. Dr. Noyes saw him five months later, and, making an exploratory incision, found the foreign body, and after great difficulty succeeded in removing it. It was four and a half inches long and one inch and a quarter thick, and had penetrated the roof of the orbit and the frontal lobe of the brain. There had not been a single symptom of any disturbance of the brain. On the fourteenth day evidences of abscess of the brain appeared, and, on enlarging the hole in the roof of the orbit, pus was found outside the dura mater and in the brain-substance. On the sixteenth day paralysis of the arm and leg on the opposite side appeared. The skull was trephined, and pus was found in the brain at a depth of one inch and three-quarters. The patient died on the thirty-ninth day.

The use of the Röntgen ray has been recently tried in cases of supposed foreign body in the orbit, and in some instances the value of repeated and differently placed exposures in determining the location of foreign bodies has been proved. This was shown by Dr. C. A. Oliver, of Philadelphia, in a paper read before the American Ophthalmological Society in 1897, the method employed having been devised by Dr. Charles L. Leonard, of Philadelphia. The method consists in making the base-line for triangulation anterior to the cranial shadow, and the exposures are repeated sufficiently often at fixed distances and set situations to give a multiple series of relational sides and angles, from which the position of the foreign body can be accurately determined. No fixed apparatus is necessary. A full account of the method will be found in the "Transactions of the American Ophthalmological Society for 1897."

INJURIES TO THE MUSCLES IN THE ORBIT.

In extensive lacerated wounds of the orbital tissue, particularly in cases where foreign bodies have entered deeply into the orbit, the ocular muscles may be directly involved in the injury; but actual division of the motor nerves must be extremely rare, for Berlin could find none such reported in literature. He himself reports two cases, which cannot be explained in any other way than by division of the nerve-fibres supplying the muscles.

Disturbances of motility, with no loss of continuity of the muscular fibres, are due to a displacement of the eyeball forward, or luxation with simultaneous mechanical obstruction to the normal muscular action, accompanied by a varying amount of exophthalmos. If, however, the muscles have been ruptured by the injury, the eyeball is usually luxated entirely outside the eyelids, which then contract behind it. It is a rare form of injury, and is usually accompanied by laceration or rupture of the optic nerve and loss of sight. In some rare cases of exophthalmos without injury, it is possible to produce extreme luxation outside of the eyelids by pressing the eyeball forward with the fingers or thumbs, owing to an

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extremely relaxed condition of the muscles. The mechanism of this luxation is of course due to some force acting from behind forward upon the eyeball and pushing it out of the orbit. This force may be of very varying nature. It may arise from a sudden enormous distention of the orbital vessels, with or without rupture and extravasation of blood. It sometimes occurs by the introduction of an elevator or speculum beneath the lids. The luxation of the eyeball may be due to sudden narrowing or obliteration of the posterior segment of the orbit, caused by fracture of the orbital walls or undue crushing of the head of an infant by forceps during an instrumental delivery.

The ocular muscles are sometimes injured without any accompanying luxation of the eyeball; but these accidents are rather rare, not more than twelve or fifteen having been reported. The internal rectus is the most frequently injured. In these cases the loss of continuity of the muscular fibres has been both partial and complete. The prognosis, so far as the muscle is concerned, is generally favorable, for if a functional loss of power result it may be restored by operative interference. If the muscle has been completely divided, it must be reattached to its tendon or to the eyeball by sutures through the muscle and conjunctiva, and this is the more readily done the more recent the injury. If the muscle has been only partially divided, it is sufficient to close the conjunctival wound by sutures.

When the eyeball has been luxated, it must be replaced between the lids as carefully as possible. If this does not succeed readily by the aid of an oiled spatula introduced beneath the upper lid while steady pressure backward is made upon the eyeball, then the external canthus must be divided, the eyeball replaced, and the canthus again stitched up. If, however, the eye is sightless and the optic nerve has been ruptured, the globe should be removed at once and the case treated as an ordinary enucleation.

Injuries of the lacrymal gland, injuries of the optic nerve within the orbit, and injuries of the eyeball will be fully treated of in other sections of this work.

EMPHYSEMA OF THE ORBIT.

Emphysema, or infiltration of air into the cellular tissue of the orbit, is merely a symptom, and not a disease. It is an important aid in enabling us to diagnose direct fracture of the inner wall of the orbit, whether involving the ethmoid, the lacrymal, or the nasal bones. It should not, however, be ignored that a communication between the orbit and certain air-spaces in the skull may also be caused by a fracture or dislocation of the inferior or superior orbital walls, opening on the one hand into the maxillary sinus and on the other into the frontal sinus, although both communicate indirectly with the sinuses of the nose and ethmoid cells. Emphysema of the orbit is usually associated with a similar condition of the eyelids. The extravasated air generally comes from the ethmoid cells. The first symptom here is exophthalmos, which is increased by forced expiration.

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The protrusion of the eye is accompanied by some displacement and by some limitation in motility, as a result of the mechanical obstruction. The exophthalmos diminishes under direct pressure upon the eyeball backward. The cause is commonly a severe injury which has fractured the orbital wall directly or indirectly, and usually the inner wall, and which has at the same time ruptured or torn through the periosteum. Any forced expiration will immediately drive the air through the fracture into the orbital tissue, and even in ordinary respiration some air is certain to pass into the orbit. In the cases reported, where emphysema of the orbit occurred during violent coughing or sneezing, without any preceding injury, the probable explanation is that some loss of continuity of the bony wall had always existed, and the first unusually violent expiratory effort produced the emphysema.

The *prognosis* is always good, and the *treatment* consists in the careful avoidance of any violent expiration and the application of a light pressure-bandage.

HEMORRHAGE INTO THE ORBIT.

Extravasations of blood within the orbit may come from ruptured blood-vessels in the orbit or from vessels outside of the orbit. They are either spontaneous or traumatic, the latter being much the more frequent. The blood may be extravasated between the periosteum and the bony wall, or in the orbital cellular tissue, or within the oculo-orbital fascia or capsule of Tenon. The latter is the seat of the hemorrhage which so frequently occurs after tenotomy for the relief of squint. When the hemorrhage occurs beneath the periosteum, it may be associated with hemorrhage in the orbital cellular tissue, and is usually due to fracture of the orbital walls or base of the skull.

Spontaneous hemorrhage into the orbit is very rare, and when it occurs is due to hæmophilia, scurvy, disease of the walls of the blood-vessels, or sudden, forced muscular action, as in violent coughing or retching. The rarity of such spontaneous hemorrhages is probably due to the uniform pressure exerted upon the vessels by the eyeball and soft contents of the orbit.

Hemorrhages into the orbit due to traumatism are much more frequent than the spontaneous variety. Actual lesions of the orbital vessels occur in all sorts of penetrating wounds of the orbit. Indirect lesions of these vessels are met with in cases of contusion or concussion of the eyeball, with rupture of capillary vessels immediately around the eyeball as a result of the blow. Hemorrhages produced in this way are generally accompanied by fracture of the bones entering into the formation of the orbit, as the result of a blow or fall upon the head.

Symptoms.—The symptoms of hemorrhage into the orbit are extravasation of blood beneath the conjunctiva and into the tissue of the eyelids (suffusion), exophthalmos, and more or less limitation of motility in the eyeball. If the exophthalmos occur suddenly or immediately after an

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injury to the orbit, the presence of an orbital hemorrhage is extremely probable; and if the protrusion of the eyeball is accompanied or followed very shortly by suffusion of the conjunctiva and eyelids, the diagnosis is certain. The exophthalmos is usually directly forward, but may be entirely absent, and here the diagnosis is aided by other signs of pressure in the orbit and by the suffusion of the conjunctiva. The protrusion of the eyeball and the suffusion of the lids and conjunctiva have long been regarded as evidences of a fracture at the base of the skull, involving the orbital walls in whole or in part; but it should not be forgotten that hemorrhage into the orbit of considerable extent may occur as the result of indirect injury, without any fracture of the orbital walls; and that, on the other hand, fracture of the bones of the orbit has been observed without any accompanying extravasation of blood in the orbit. In the former case the hemorrhage has undoubtedly come from the vessels of the orbital tissue. Hemorrhage into the orbit after blows or falls upon the head has, however, prognostic significance as a sign of the presence of a serious injury; and in this sense the hemorrhage is of great symptomatic value, even in those cases in which there is no fracture of the roof of the orbit.

Sometimes the extravasation of blood in the orbit does not extend beneath the conjunctiva and into the lids, but runs down into the nose and pharynx. This would indicate not only the laceration of some large blood-vessel in the orbit or its immediate vicinity, but also the existence of a communication between the orbit and the cavities of the nose or pharynx, and it might be so profuse as to endanger life and require ligation of the carotid artery. The existence of a large hemorrhage in the orbit would also not only hinder the free motility of the eye, but cause impairment of vision by pressure on the optic nerve.

The usual course of an orbital hemorrhage ends in absorption of the extravasated blood in from three to six weeks, and in complete restoration of all the functions of the eyeball. If, however, the hemorrhage has been very extensive and the protrusion of the eye excessive, so that the lids cannot be closed over it, the globe may be destroyed by neuro-paralytic keratitis and abscess of the cornea.

Treatment.—Slight hemorrhages into the orbit usually disappear spontaneously. The application of iced compresses and a firm bandage generally suffice for the more serious cases. If there is much pressure upon the eyeball and marked exophthalmos, the blood may be let out through a free opening with a broad bistoury; but this is rarely necessary, and should always be done under the strictest antiseptic precautions.

ORBITAL CELLULITIS, OR PHLEGMON OF THE ORBIT.

This is an inflammation of the fatty cellular tissue of the orbit, and may be acute, subacute, or chronic in character. It may be confined to one side or involve both orbits. In favorable cases it may undergo resolution and

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disappear without leaving any lasting trace of its presence, but in the great majority of cases it ends in suppuration and the formation of an abscess.

Etiology.—It may be idiopathic or traumatic in origin. When of traumatic origin, orbital cellulitis may be due to fracture of one or more of the bones entering into the formation of the orbit; to penetrating wounds of the orbital tissue, with or without the entrance and lodgement of foreign bodies in the orbit; or to surgical operations involving the contents of the orbital cavity. The idiopathic causes are: 1, long-continued exposure to cold; 2, periostitis, whether of syphilitic, scrofulous, rheumatic, or gouty origin; 3, the exanthematous fevers, especially scarlatina and typhoid; 4, meningitis through the medium of thrombosis of the cavernous sinus and ophthalmic veins; 5, facial erysipelas; 6, extension of the inflammatory process from diseased teeth in the upper jaw; 7, suppuration in the ethmoid cells or sphenoidal sinus; 8, metastatic inflammation due to general pyæmia or puerperal septicæmia; 9, panophthalmitis through the medium of inflammation of the capsule of Tenon; 10, in very rare instances, inflammation in and around the lacrymal gland.

In orbital cellulitis following periostitis, the attack may be either acute or chronic, and there is almost always more or less extensive caries of the bone present. It is usually unilateral, and the suppuration generally continues until the cause is removed and the carious bone is cast off. In cellulitis occurring in the course of the exanthemata or meningitis, the disease is almost always confined to one side; but when due to facial erysipelas, both orbits are usually involved, the symptoms are of the most violent character, and the results are most destructive not only to the integrity of the eyeball, but very often to life.

Symptoms.—In the mild subacute and chronic forms of the inflammation, there is dull pain in and around the orbit, slight swelling of the lids without much discoloration, and slight divergence or protrusion of the eye with diplopia, but without the presence of the usual inflammatory symptoms or constitutional disturbance. These cases usually end in resolution without the need of surgical interference.

In the acute form the symptoms are all more violent. The attack is usually ushered in by a chill, followed by fever; severe deep-seated pain in the orbit; general headache; limitation in motility of the eyeball, often amounting to complete immobility; swelling and œdema of the lids, accompanied by a marked dusky hue of the skin, simulating a case of acute blennorrhœa of the conjunctiva; hyperæmia and chemosis of the ocular conjunctiva; exophthalmos; defective vision due to neuro-retinitis, with exudation and hemorrhages in the retina; anæsthesia, ulceration and suppuration of the cornea, and panophthalmitis. On digital examination, all the tissues of the orbit are found tense, hard, and resisting, and the slightest pressure causes severe pain, especially when made above the eyeball, beneath the supra-orbital margin. The severer symptoms are always present in cellulitis following erysipelas, and when the cornea remains clear the optic

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nerve is seen to be swollen, pale, and infiltrated, and the vessels are much reduced in calibre. In these cases thrombosis of the orbital vessels is almost sure to occur. Ulceration and suppuration of the cornea are more likely to be met with if the chief severity of the inflammation lies anterior to the equatorial region; while the exophthalmos and neuro-retinitis are more marked if the inflammation is mainly centred back of the eyeball, towards the apex of the orbit. If the optic nerve be affected far back at the apex, there may be no ophthalmoscopic evidence of any nerve-trouble. If the pressure affect the nerve immediately behind the eye, there will be the usual signs of optic neuritis. If the point of attack be farther back, there may be at first merely the signs of retro-bulbar neuritis. In nearly all cases in which the optic nerve is involved, more or less complete atrophy of the nerve follows.

Course and Prognosis.—In mild cases the prognosis is favorable, and, unless complicated by caries of the bony wall, the disease is short in duration. In severe cases of the acute type the prognosis is very unfavorable, and if both orbits are involved, as so frequently occurs in erysipelas, the disease is generally destructive to the eyes and very often to life, by reason of the extension of the inflammatory process backward through the optic foramen and sphenoidal fissure, or by the production of general pyæmia. Phlebitis of the orbital veins may extend to the brain through the cavernous sinus, and end in encephalitis. In rare cases of unilateral cellulitis the disease has been known to pass to the cavernous sinus on the opposite side and involve the second orbit by what may be called an ascending phlebitis.

Treatment.—This will vary according to the type of inflammation and the stage at which it is first seen. If the case be one of a mild type, with little or no constitutional disturbance, an application of two or three leeches to the brow or temples should be made, followed by frequently changed hot compresses to the closed lids and surrounding region. At the same time iron and quinine may be administered, if necessary. If, on the contrary, the case be one of the acute phlegmonous type, the treatment must correspond. Six or eight leeches should be applied to the brow or temples, and the hot compresses must be continually employed and rapidly changed. If the swelling of the lids is marked, the infiltration of the orbit tense, and exophthalmos present, with much pain, it is not advisable to wait for the orbital abscess to point, but free incisions must be made into the orbital tissue through the conjunctiva. These incisions should be made with a straight, narrow bistoury at the point of greatest tension, close to and parallel with the wall of the orbit, and the knife should be introduced for an inch or more towards the apex of the orbit, carefully avoiding the eyeball. The external opening of the incisions should be freely enlarged. If the lids cannot be opened so as to reach the conjunctiva, the incisions must be made through the skin of the upper lid, just beneath the superior orbital margin. These cuts relieve the tension of the oculo-orbital fascia, unload the surcharged vessels, and relax the tissues. An early incision and a suffi-

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ciently large external opening will sometimes aid in arresting the inflammation and prevent the formation of pus, and in any event will tend to allay the disastrous effects of pressure on the optic nerve and eyeball, and may prevent ulceration and sloughing of the cornea, ending in panophthalmitis. It sometimes is necessary to make more than one incision in different parts of the orbit. As soon as the incision has been made, it must be thoroughly irrigated with a warm solution of mercuric bichloride (1 to 2000), and the wound must be kept open with a plug of antiseptic lint or cotton. These irrigations must be repeated several times a day, and in the interval the hot applications to the lids must be continued. In this way local bleeding and subsequent purulent discharge are fostered, and at the same time the suppuration is limited in extent and shortened in duration. There is comparatively little danger from excessive hemorrhages in these cases, as the vessels are more or less choked. When the cause has been facial erysipelas, incisions must be made early, not only in the orbit but in the lids, as they afford the best method of preventing extensive sloughing of the tissues. The patient's general condition will modify this local treatment, and may necessitate the use of stimulants, food at short intervals, quinine, acids, and opiates. If the cornea become involved early in the disease, paracentesis or its free division may be required. If extensive abscess of the cornea or panophthalmitis with increased tension be present, it may be necessary to divide completely the anterior segment of the eyeball and evacuate its contents. This seems a better and safer procedure than enucleation of the eyeball, for meningitis and death have sometimes occurred in such cases, owing to the very free opening into the lymphatic spaces around the eyeball and the intervaginal lymph-channel along the optic nerve, thus possibly facilitating the progress of the bacteria to the intra-cranial cavity. The mode of propagation to the brain has not, however, been clearly made out in these cases. The same objection does not apply to enucleation in cases of primary panophthalmitis without coexisting orbital cellulitis. Evisceration or exenteration of the eyeball is probably a less severe operation than enucleation, and therefore to be preferred when the patient is old or enfeebled. The hot applications and free irrigation should be kept up as long as there is any purulent discharge or any tense swelling in the orbit. Subsequently, when all inflammatory symptoms have subsided, the enucleation of the more or less atrophied stump may be done, and thus an additional source of irritation be removed.

Evisceration of the eyeball, followed by the insertion of an artificial vitreous or glass globe, is a much better operation in these cases than enucleation. The method is that devised by Mules. The conjunctiva should be dissected for some distance around the cornea; the cornea should then be removed, together with a triangular portion of the sclerotic above and below. The contents of the eye are then removed with a Volkmann spoon and the cavity irrigated with a solution of mercuric bichloride (1 to 3000). The glass globe is then inserted and the scleral aperture closed

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vertically by four or five white silk sutures passed through the entire thickness of the sclera and allowed to remain permanently. The conjunctiva is then united transversely by three or four black silk sutures, which are subsequently removed. All the steps of the operation are done under the strictest antiseptic precautions.

INFLAMMATION OF THE CAPSULE OF TENON, OR OCULO-ORBITAL FASCIA.

In many forms of retro-bulbar inflammation the connective tissue which enters into the formation of the fibrous capsule of the eyeball, and which is intimately connected with the retro-bulbar adipose tissue, becomes more or less involved in the inflammatory process; hence in all cases of pronounced orbital cellulitis or panophthalmitis the capsule of Tenon is also the seat of inflammation and infiltration.

The oculo-orbital fascia may, however, be attacked by an inflammatory process primarily and independently, without the coexistence of orbital cellulitis. The *symptoms* of a well-marked case of inflammation of the capsule of Tenon are swelling and œdema of the upper lid, mainly confined to the upper or retro-tarsal portion; pain on the slightest movement of the eyeball; slight exophthalmos, generally straight forward; limitation in motility of the eyeball; and chemosis more or less marked of the ocular conjunctiva. This may be confined to a small area, and looks like a vesicle situated usually over the insertion of the tendon of one of the straight muscles of the eye, or it may extend all around the anterior segment of the globe. The media of the eye at first are clear and the fundus is normal. It may be either idiopathic or traumatic in origin, and the nature of the affection varies according to its origin. The idiopathic form is almost always of a rheumatic or gouty nature, is mild in type, and the exudation is of a serous character. The prognosis is good, though the duration of the disease may be prolonged to weeks before resolution is established. The traumatic form is due sometimes to accidents like penetrating wounds, but is generally the result of some surgical operation, notably tenotomy or advancement of a muscle for the cure of strabismus, and quite a number of such cases have been published. The inflammatory process is here almost always a suppurative one, and the prognosis is always grave. The writer has known of a case in which simultaneous tenotomy of both internal recti muscles was followed by purulent inflammation and loss of both eyes.

Treatment.—In the idiopathic form, the treatment should consist of hot fomentations to the closed lids, more or less continuously applied according to the severity of the attack; anodynes to relieve pain; and the internal administration of potassium iodide or sodium salicylate. In the traumatic form, the hot fomentations should be of mercuric bichloride (1 to 5000), and the culs-de-sac should also be frequently irrigated with the same solution. Any signs of the formation of pus must be followed immediately by

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free incisions into the capsule at the point indicated, and, if necessary, the incisions should be carried well back into the orbital cellular tissue. The wounds should then be frequently irrigated with an antiseptic solution and free drainage maintained. If general orbital cellulitis can be prevented, the case will recover, with restoration, at least in part, of the functions of the eye; but if cellulitis be developed, the eyeball will probably be lost by panophthalmitis.

THROMBOSIS OF THE ORBITAL VEINS AND CAVERNOUS SINUS.

In phlegmonous inflammation of the orbit, thrombosis of the orbital veins almost of necessity occurs, and is recognized as an ordinary complication of orbital cellulitis. Pure, uncomplicated thrombosis of the orbital veins, without any demonstrable cause, is difficult to distinguish from phlegmonous cellulitis of the orbit, as similar symptoms occur in both diseases. If, however, cerebral symptoms manifest themselves, these prove that the cavernous sinus has become involved in the process.

Thrombosis of the cavernous sinus is not, strictly speaking, a disease of the orbit, but it produces so many orbital symptoms that it seems best to consider it here. The thrombotic process may extend from the orbital veins to the sinus, and thence to other connecting sinuses, and give rise to recognized cerebral symptoms. If the process extends from one cavernous sinus, through the medium of the sinus circularis, to the cavernous sinus of the opposite side, there will result obstruction to the venous circulation in both orbits simultaneously, bilateral exophthalmos, œdema of the lids and face, grave cerebral symptoms, and usually death. It is a very fatal and, fortunately, a rare affection. The symptoms are more or less pronounced exophthalmos; paralysis of one or more of the ocular muscles from pressure on their nerves, or the development of small abscesses in the muscular tissue; œdema of the conjunctiva and eyelids, sometimes extending down upon the cheek or upon the temple; more or less immobility of the eye; intense pain in the ophthalmic branch of the fifth nerve, including the supra-orbital, supra-trochlear, and nasal terminal filaments, followed in many cases by anæsthesia of the same nerves from pressure; mydriasis; engorgement of the retinal veins, and even papillitis; impaired vision; anæsthesia of the cornea, and possibly corneal abscess, from impaired nutrition; œdema of the mastoid region; delirium, coma, and death. The retinal engorgement, like the exophthalmos, is due to the extreme degree of venous stasis which exists in these cases. The œdema of the mastoid region is also due to the same cause. In this region the emissary vein of Santorini empties into the transverse sinus and thus indirectly into the cavernous sinus, so that occlusion of the latter sinus would indirectly produce venous stasis in the mastoid region. Whenever this mastoid œdema is present, it forms an important diagnostic sign between thrombosis of the sinus and retro-bulbar

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cellulitis, for it is never present in the latter disease. If the thrombosis be of a septic character, whether as a result of local or of general infection, small abscesses may be developed in the eyelids and vicinity, and death usually results from meningitis, abscess of the brain, or pyæmia.

The *prognosis* is very unfavorable, most cases terminating fatally.

The *causes* of thrombosis of the cavernous sinus are, according to Berlin, marasmic conditions, infectious diseases by metastasis, diseases in the vicinity of the sinus, such as caries of the petrous bone or abscesses at the roots of the teeth, compression of the sinus or of the veins of the neck in its vicinity, chronic suppurative otitis media with extensive disease of the petrous bone or other bones of the skull, facial erysipelas, boils on the face, purulent deposits about the head, scarlatina, etc.

ENOPHTHALMOS.

Enophthalmos, or retraction of the eyeball within the orbit, is a condition which has occasionally been observed. It may be of idiopathic or of traumatic origin. Von Graefe observed a decided backward sinking of the eyeball in cholera patients to such a degree that in some cases the upper lid actually curved backward. The sinking of the eyeball occasionally observed in exhausting diseases is perhaps more seeming than real, though it may be an actual recession of the eye due to absorption of the retro-bulbar fatty tissue of the orbit. Björnström has described a variety of enophthalmos occurring periodically with neuralgia of the fifth pair of nerves; and the displacement backward of the eyeball which has been observed in cases of paralysis of the sympathetic nerve is probably identical with this form. Enophthalmos of traumatic origin is met with in cases of fracture of the bones of the orbit, and has been known to follow the injury immediately. When the sinking backward occurs later, the retraction is probably due to the influence of cicatrizing bands in the orbit. The latter condition may result from a chronic cellulitis of a low grade, as well as from operative procedures in the deeper parts of the orbit, such as the removal of tumors. Traumatic enophthalmos, according to Lang, is due to indirect fracture of the floor of the orbit, which is driven into the antrum; as a result of this the orbital space is enlarged, and the eyeball is pushed back by the pressure of the external atmosphere. In all cases of enophthalmos the interpalpebral fissure is narrowed, and in many of them there is myosis. Enophthalmos also occurs in neurotic facial atrophy.

PULSATING EXOPHTHALMOS.

Pulsating exophthalmos is a diseased process which is characterized by a peculiar group of symptoms, as follows: 1. Protrusion of the eyeball forward, and usually a little downward and outward. 2. The presence of peculiar audible noises over the region of the orbit and also over a more or

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less extensive region of the skull. 3. A distinct pulsation demonstrable on the eyeball or over any spot or region of the anterior orbital aperture. This complex group of symptoms is due to a lesion situated either in the orbit itself or in the cavity of the skull. If the lesion be in the orbit, it may be a true aneurism or a traumatic or spurious aneurism. The latter may be diffuse or circumscribed, or it may communicate with both artery and vein and form an arterio-venous or varicose aneurism. Some of the rarer lesions producing the symptoms of pulsating exophthalmos are aneurisms by anastomosis and true angiomata or erectile tumors. If the lesion be situated in the cavity of the skull, it may be an aneurism of the ophthalmic artery at its origin from the internal carotid, or an aneurism of the carotid artery itself, or the formation of a so-called arterio-venous aneurism by a rupture of the internal carotid artery in the cavernous sinus.

Symptomatology.—The symptom first noticed by the observer is the exophthalmos, which is generally unilateral and of considerable degree. Very often the protrusion is so great that the lids cannot be closed over the eye. The protrusion of the eye is forward and downward-outward or downward-inward. The upper lid is usually swollen and tense, and the skin is livid-red and shining, the veins being enormously swollen and the temperature above normal. The tarso-orbital fold is lost, and in rare cases the upper lid is everted. The lower lid is frequently everted, and the palpebral conjunctiva greatly swollen and prominent. The ocular conjunctiva is chemotic, and the vessels are enormously distended and dark red in color. If the exophthalmos be marked, the cornea becomes dull and slightly cloudy, readily ulcerates, and loses its sensibility. The iris becomes very hyperæmic and dilated, and acts very sluggishly. The protruding eyeball can readily be replaced in the orbit by pressure, but the exophthalmos immediately returns when the backward pressure is relaxed. The pulsation of the eyeball is sometimes visible to the eye as well as perceptible to the touch, and is rhythmical with the pulse. In addition to the pulsation, the fingers when laid upon the eyeball will feel a more or less distinct thrill.

Another important symptom is the presence of a pulsating tumor close to the eyeball, which is not always demonstrable. Its usual location is upward and inward, between the eye and the upper margin of the orbit. The tumor is soft, very compressible, and gives a distinct thrill to the finger. If the ear or the stethoscope be applied to the eyeball or to the upper margin of the orbit, a more or less distinct blowing noise is heard, which at first appears to be intermittent, but closer observation shows that it is merely an intensified continuous soughing or murmuring. Sometimes the sound changes markedly in pitch and becomes almost whistling. By compression of the common carotid artery in the neck on the corresponding sides, the pulsation and noise either cease immediately and entirely, or become very much less marked.

If the cornea be clear, the ophthalmoscopic examination frequently

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reveals a typical case of papillitis, with the veins increased enormously in diameter and curiously distorted and presenting numerous varicose dilatations. Venous pulsation is always very marked, and arterial pulsation on the disk is occasionally met with. The retinal arteries are reduced in calibre and sometimes appear like mere threads. Striated and punctate hemorrhages are also often present in the retina. These ophthalmoscopic symptoms are, however, by no means always present. In cases with marked deterioration of vision, the optic disk appears discolored and of a dirty yellow, with engorged veins and attenuated arteries.

Where the pulsation of the eyeball is marked, a rhythmical movement in the papilla synchronous with the cardiac systole has been observed.

The media of the eye, except the cornea, are usually transparent.

The motility of the upper lid and of the eyeball is decidedly limited in almost every case. The pupil is dilated and usually immovable. The accommodation is diminished or entirely lost, and the refraction of the eye is sometimes lessened by pressure from behind upon the eyeball. The condition of the vision varies greatly. In the great majority of instances it either remains intact throughout or is but slightly affected. In a small minority of cases the vision seems to be seriously affected from the beginning of the disease, and in some few cases is rapidly and permanently destroyed.

In addition to the disturbance of the vision, we sometimes meet with diminution or entire loss of sensibility in the region of the first branch of the trifacial nerve, paralysis of the facial nerve, and certain anomalies in the sense of hearing.

The *subjective* symptoms are generally very annoying. There is often severe pain in the orbit and head from the very beginning, and, though it may subside and even entirely disappear, it is usually present till the end. The patients complain of a knocking, roaring, or buzzing in the head and ears, which is even more annoying than the pain. Vertigo is also mentioned, as well as a constant feeling of heat in the head.

Etiology and Course of the Disease.—For convenience the cases of pulsating exophthalmos may be divided into two classes, spontaneous or idiopathic and traumatic. In the idiopathic cases the appearance of the symptoms in the great majority of cases is very sudden. Without any warning, the patient is seized with a sudden severe pain in the head or eyeball, or hears a sudden noise like a pistol-shot in his ear, accompanied by a feeling as if something had burst inside the orbit or skull. This is followed by a buzzing or roaring in the head, which rapidly increases in intensity and never ceases. After a few hours or, at most, days, the lid begins to swell, the conjunctiva becomes chemotic, and all the other symptoms rapidly make their appearance.

In the traumatic cases, or at least in the great majority of them, we must look for an injury which from its very nature is likely to have produced a fracture at the base of the skull. The patient may be found un-

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conscious and bleeding from one or both ears as well as from the nose and mouth. The bleeding from the ear may be, and usually is, considerable, and lasts for several hours. Sometimes the patient is hemiplegic on the side corresponding to the bleeding from the ear. In some cases there appear immediately, or after the lapse of a few hours, a subconjunctival hemorrhage and ecchymosis of the lids, which point to hemorrhage into the orbital tissue and fracture of the roof of the orbit. The exophthalmos and the pulsation next follow with tolerable rapidity, and if both orbits are involved in the injury, the protrusion occurs sooner in one orbit than in the other. In the great majority of cases all the important symptoms are developed within the first two months. In some rare cases, however, the progress of the disease is very insidious, and the objective symptoms manifest themselves only after a number of months have elapsed.

In a certain number of cases hemorrhages occur repeatedly, and of so threatening a character that ligation of the common carotid becomes necessary in order to save the life of the patient. They usually come from the nose, though they have been known to come directly from the tumor through a rupture in the conjunctiva.

A few cases have been put on record in which spontaneous resolution and disappearance of the tumor and subsidence of all the symptoms have occurred. There are also cases which have been apparently healed by dietetic and medicinal treatment, but they form a small minority of the cases that apply for treatment. The idiopathic cases seem to occur mainly in the female sex, for of thirty-two such cases reported by Sattler, twenty-three occurred in women and only six in men. In three cases the sex was not mentioned.

Most of the cases of idiopathic pulsating exophthalmos occur between the thirtieth and fiftieth years of age.

Traumatic pulsating exophthalmos is met with much more frequently among men than among women, which is what might be expected from the more dangerous nature of men's occupations.

Pathology.—The true pathology of pulsating exophthalmos is but little understood. An autopsy has been made in only a very limited number of the published cases, and the results of such examination were so various that the true nature of the lesion cannot be exactly determined in every case. Aneurism of the ophthalmic artery within the orbit has twice been found in the cadaver.

Nunneley reports a case of aneurism of the ophthalmic artery posterior to the orbit in a patient upon whom the common carotid artery had been ligated five years previously for spontaneous pulsating exophthalmos. In a number of cases of traumatic origin the autopsy revealed laceration of the internal carotid within the cavernous sinus. Spontaneous rupture of an aneurism of the internal carotid within the cavernous sinus has also been found. Another post-mortem result was inflammation and thrombosis of the cavernous, circular, and transverse sinuses and the ophthalmic veins,

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with purulent softening of the thrombi. The most exhaustive account of the subject is given by Sattler.[1] Of eighteen autopsies reported since the publication of Sattler's article, in seven there was no aneurism found. Of the eleven remaining cases, aneurism of the ophthalmic artery in the orbit was found in two cases, aneurism of the same artery before its entrance into the orbit was found in one case, in four cases of traumatic origin there was an aneurism of the carotid artery in the cavernous sinus, in three cases the carotid aneurism had ruptured in the cavernous sinus, and in one case there was spontaneous dilatation of the carotid artery in the cavernous sinus.

Diagnosis and Prognosis.—It is always a difficult matter to distinguish between the different varieties of pulsating tumors in the orbit, and in many of them it is well-nigh impossible. The first and most important point for us to determine, if we can, is whether the pulsating exophthalmos in a given case depends on a benign or malignant tumor, or on an aneurism, or on inflammation of the sinuses. Vascular tumors, as a rule, are more resistant to pressure than aneurisms, and their position in the orbit is usually out of the line of the axis. Their development is much slower than that of an aneurism. Malignant tumors of the orbit with pulsation and protrusion of the eye are generally of rapid growth, and are apt to be accompanied by local hemorrhages, metastatic growths, and a general cachectic condition. Pulsating angiomata are less painful than malignant tumors, and tend to expand more easily after removal of pressure.

Another important point in diagnosis is to determine the possible location of an aneurism, whether in the orbit or posterior to the orbit in the sinus. We may have both true and false aneurisms of the ophthalmic artery. A diffuse false aneurism in the orbit may be due to laceration of the ophthalmic artery in the optic foramen by a fracture of the wall of the canal. Aneurism by anastomosis, or the so-called cirsoid aneurism, has never yet been found in the orbit, though its presence has often been assumed. Mixed tumors, partly angiomatous and partly aneurismal, have been met with in the orbit as in other regions of the body. The exact mode of origin of these mixed forms is not positively known, but it is probable that they are developed in the same way as is seen in the transformation of simple, non-pulsating nævi in the skull and face into pulsating vascular tumors, and one such case has been reported by Frothingham. The plexiform angiomata occasionally met with in the orbit are essentially venous in character, consisting of a convoluted mass of distended veins held together by loose connective tissue. The congenital variety of pulsating angiomata may be confounded with a congenital encephalocele, and here the differential diagnosis must be based mainly upon the objective symptoms. Another form of vascular tumor in the orbit which may be confounded with true pulsating exophthalmos is a varicose dilatation of the

[1] Graefe-Saemisch, Handbuch der gesammten Augenheilkunde, vi. 745–943.

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ophthalmic veins and their branches, but the combined symptoms are quite different. In the latter condition there are no pulsation and no bruit, and none of the usual symptoms of stasis in the orbit and eyeball.

Still another form of vascular tumor met with in the orbit is the pulsating encephaloid tumor,—a very rare condition. When a soft orbital tumor is found to be very vascular, with numerous large arteries running through, which give it a decided pulsating character, it is almost certain to be malignant and sarcomatous in character. Such a tumor is apt to be very soft, almost fluctuating in character, and with a smooth surface, and may be frequently mistaken for a true pulsating exophthalmos. But such a tumor does not yield as completely under the pressure of the fingers as a true pulsating tumor does. Another point of differential diagnosis is the position of the pulsating tumor, the true pulsating exophthalmos being more frequently found at the upper and inner portion of the orbit. Multiple pulsating growths in and about the orbit would indicate that they were malignant in character.

When we come to consider pathological conditions posterior to the orbit, associated with pulsating exophthalmos, the subject becomes more complicated. True aneurism of the ophthalmic artery within the cavity of the skull is one of the rarest causes of pulsating exophthalmos, and then only when it has been developed very *rapidly;* for when such an aneurism has been developed *gradually* to such an extent as to compress the calibre of the ophthalmic vein and cavernous sinus, all signs of stasis and pulsation in the orbit may be entirely absent, owing to the development of a collateral circulation. This opinion receives corroboration from a study of the cases of true aneurism of the internal carotid within the cavernous sinus. In none of the cases which came to autopsy were there any signs of stasis, exophthalmos, or pulsation in the orbit during life. Most of the cases of pulsating exophthalmos consequent on some extra-orbital lesion have been found to be due to a rupture of the internal carotid within the cavernous sinus. The rupture may be of spontaneous or traumatic origin. If spontaneous, the rupture has occurred either in an artery already the seat of an aneurism, or in a vessel with abnormally thin and diseased walls. If traumatic, the arterial wall may have been either directly or indirectly torn as the result of injury. The traumatism may be either by means of a foreign body like a bullet, or by a splinter of bone from a fractured skull. The foreign body may enter the orbit of one side, pass through the sphenoidal sinus, and wound the cavernous sinus on the other side, or it may follow the orbital wall to the apex and involve the cavernous sinus and internal carotid artery of the same side. Indirect traumatism is much the more common cause. Here we have to do with a more or less extensive fracture of the base of the skull and the probable laceration of the cavernous sinus and internal carotid by a sharp splinter of bone. In those rare cases in which the sinus and artery have been lacerated by a fall or blow upon the head without any fracture at the base of the skull, there must have existed some diseased condition

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of the walls of the vessels which predisposed to rupture. The immediate consequence of a rupture of the carotid artery in the cavernous sinus is an increase in the venous pressure in the latter by the influx of arterial blood, the degree of which depends upon the extent of the rupture in the arterial wall. The signs of stasis are first seen in the superior ophthalmic vein. After a varying time the arterial pulsation is gradually propagated to the veins, and then the first pulsation is felt, and usually in the upper portion of the orbit or at the inner canthus. The signs of stasis next are met with in the retinal veins, and here also pulsation of the veins makes its appearance later. The same distended condition rapidly occurs in the meningeal and cerebral veins, which is the cause of the violent pain in the head so constantly complained of by the patients. The bruit which in many cases is so marked an objective symptom is a continuous murmur or buzzing sound, which with every systolic action of the heart is complicated by a vesicular murmur. The subjective noises are excessively annoying to the patient, but of much less consequence from the diagnostic standpoint.

One of the early symptoms of rupture of the carotid in the sinus is paresis of one of the motor nerves of the eye and of the first branch of the fifth nerve. The optic nerve is not at first affected by rupture of the carotid, and sometimes the vision remains intact throughout. In those cases in which the sight is early and seriously affected, the impairment of vision is due to the very marked condition of venous stasis. Blindness *may* come on immediately after the occurrence of the injury, and is then almost always due to a simultaneous laceration or rupture of the optic nerve. As a remote effect of such extreme injury, ulceration and perforation of the cornea and irido-choroiditis are occasionally met with. The former of these complications is due to paralysis of the fifth nerve.

Those cases of pulsating exophthalmos due to rupture of the internal carotid within the cavernous sinus may be divided into two classes,—viz., 1, those in which all the symptoms are fully developed within twenty-four hours of the receipt of the injury; and, 2, those in which months elapse before all the symptoms are developed. The latter class is much the more numerous. Anything which will increase the vascular tension within the carotid will tend to the rapid development of all the symptoms, and change a gradually developing pathological process into an acutely rapid one. Dead-house investigations have demonstrated the fact that the internal carotid is much more predisposed to the development of aneurismal dilatation than any other artery of a corresponding size. One of the remote contingencies which might have a bearing on the general subject is whether a neoplasm which has developed in the cavernous sinus, pressed upon the internal carotid, and extended into the orbit, might not give rise to the symptoms of pulsating exophthalmos by obstructing the return venous current through the superior orbital fissure. Nunneley believes this possible, mainly on theoretical grounds. Sattler thinks that such symptoms might

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be induced if the tumor in the sinus were small, and if a fold of dura mater were pushed forward into the orbit between the surface of the tumor and the eroded hole in the orbit, so that the pulsation of the artery could be communicated through the medium of the tumor and the meningeal fluid to the fingers of the observer.

In all these cases the probability of a spontaneous cure is slight, and when such a cure does take place it is almost always at the expense of loss of sight. The prognosis as to life is not so unfavorable, as when death results it is generally from some complication. In idiopathic cases the complication most expected is apoplexy or some other serious brain-lesion from disease of the vascular walls. In traumatic cases the great danger is the occurrence of profuse and repeated hemorrhages from the cavernous sinus, caused by fracture of the bones at the base of the skull, involving the sphenoid bone and opening the sinus. Encephaloid tumors are of the most unfavorable type, and always endanger life. Pulsating angiomata grow slowly, and do not directly endanger life, unless of great size.

Treatment.—This will vary according to the diagnosis of the existing lesion and the nature of the original one, whether spontaneous or traumatic. The two chief surgical methods of treatment are ligation of the common carotid artery and compression of the same artery. This compression may be either instrumental or digital, the latter being the more frequently employed. Sattler believes that digital compression possesses the greater advantages, for not only are the dangers which usually accompany the operation of ligation avoided, but time is also given for the complete development of the collateral circulation, even if subsequently ligation becomes necessary. Moreover, in the majority of the cases cured by compression the successful result has begun to appear on the third day, and in some has then been completely obtained. Gioppi and Scaramuzza first reported cases of orbital aneurism cured by digital compression of the carotid. In Gioppi's case the pulsation and bruit disappeared in four days, and the sight returned on the sixth day. Scaramuzza's case was cured on the eighteenth day. In the traumatic cases the chances of successful result by intermittent digital compression are not very great. The walls of the ruptured artery, except at the point of rupture, are generally healthy; its internal coat is smooth, and the conditions are very unfavorable for coagulation of blood in the artery. If the laceration is extensive, it is highly improbable that an intermittent compression lasting from five to twenty minutes would produce a clot of sufficient firmness to resist the force of the blood-current when the compression was discontinued. In the idiopathic cases the conditions are more favorable, for the arterial wall is almost always diseased, and frequently is the seat of aneurismal dilatation. If lasting success is to be obtained in the traumatic cases by digital compression, the compression must be continued uninterruptedly from three to six hours, and this is extremely difficult, on account of the position of the carotid artery in the neck, and in a short, thick neck is impossible. The compression of the

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artery against the vertebral column is best done at the level of the top of the thyroid cartilage, at the anterior edge of the sterno-cleido-mastoid muscle. A docile patient can often make digital compression on his own artery better than any one can do it for him, and is, moreover, a better judge of the correct carrying out of the pressure, by the disappearance of the subjective noises. Compression by means of instruments has been frequently tried as a means of relieving or doing away with the strict requirements of digital compression, but such attempts have generally been soon abandoned. It seems better in every case of pulsating exophthalmos to begin the treatment by the method of compression, and this at first must of necessity be intermittent. In idiopathic cases this compression will often alone suffice to effect a cure. In traumatic cases the pressure must be continuous and kept up for a number of hours. If the latter does not succeed, the way is prepared for a favorable result from ligation of the vessel by the establishment of a collateral circulation.

Ligation of the carotid is the oldest operation for the relief of pulsating exophthalmos, and has given the most satisfactory results. As a rule, the pulsation and bruit disappear immediately, any existing tumor collapses, the eye often sinks back into the orbit, and the tension and distention of the veins in the lids diminish. Faint subjective sounds may return after a few hours, but the other symptoms soon entirely disappear. The swelling of the lids and the chemosis vanish, the exophthalmos lessens, the motility of the eyeball returns, and vision is gradually restored. The swelling and cloudiness of the optic disk subside, the retinal veins become less engorged and tortuous, and the hemorrhages are absorbed. Paresis of one or more of the ocular muscles sometimes remains for a long period unchanged. In most of the cases the cure is complete in six weeks.

In a certain number of cases the symptoms returned after ligation of the carotid, sometimes on the same side and more rarely on the opposite side. In these cases it has been advised to ligate the external carotid of the same side or the common carotid of the opposite side, and successful cases have been reported of both these methods of operating. Those cases in which after apparently a complete cure the symptoms return in the orbit of the opposite side are very rare and interesting anomalies. Here the superior ophthalmic vein of the side first affected has been obliterated at its opening into the cavernous sinus by the thrombosis of the sinus produced by the ligation of the carotid. The cavernous sinus subsequently regains its permeability, and the still yielding cicatrix in the arterial wall yields and opens again. The arterial blood again enters the sinus, passes through the circular sinus into the cavernous sinus of the opposite side, and produces here the symptoms of pulsating exophthalmos.

The dangers attached to the operation of ligation of the carotid are never to be lost sight of. They consist of disturbances in the cerebral functions, secondary hemorrhage, and septicæmia from accidental wound-poisoning. Wyeth reports 43.16 per cent. of deaths in a series of seven hundred

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and eighty-nine cases of ligation of the carotid for various diseases. In sixty-three cases of ligation of the artery for pulsating exophthalmos, disturbances of the cerebral functions, such as paralysis, convulsions, stupor, etc., were observed in only four cases, and three of these ended fatally. Secondary hemorrhage may occur between the ninth and the twenty-second day, and a number of cases of death from secondary hemorrhage have been reported after ligation of the carotid for pulsating exophthalmos. The third danger—that from septicæmia—has been reduced to a minimum by the modern antiseptic methods of surgery, and causes the surgeon of the present day little or no anxiety.

In *pulsating malignant tumors* ligation of the common carotid for the purpose of cutting off their nutrient supply has not proved successful in a single recorded case, and has been entirely abandoned.

In *pulsating angiomata* some successful results have been reported after ligation of the carotid, but ophthalmic surgeons have come to prefer a local treatment or complete extirpation of the tumor.

Attempts have frequently been made to bring about coagulation of the blood in pulsating orbital aneurisms by acupuncture, galvano-puncture, and the injection of coagulating fluids of various kinds. The first of these methods has been found not only useless, but positively harmful. Galvano-puncture seems to deserve a further trial, especially in cases of congenital nævus of the lids and orbit, in angiomata, and in cirsoid aneurism. When properly done, it is perfectly safe, and might perhaps be essayed even in cases of pulsating exophthalmos, in connection with digital compression.

The subcutaneous injection of a solution of ergotin in alcohol and glycerin in the immediate vicinity of the pulsating tumor, as recommended by Von Langenbeck, has been tried in a number of reported cases, but without any beneficial result. Injections of some coagulating fluid into the interior of the pulsating tumor have also been recommended. They are more effective in their action, but much more dangerous, on account of the violent reaction, severe headaches, chills, persistent vomiting, slowing of the pulse, and sometimes panophthalmitis. Of late years this method has been entirely given up.

In the rare cases in which the presence of an aneurism within the orbit has been positively diagnosed, especially if it is far forward in the orbit, it might be well to ligate not only the vessels which enter the aneurism behind, but also those which make their exit in front, and then dissect out the sac entirely.

In pulsating angioma it is advisable to dissect out and extirpate the entire tumor, rather than to depend upon producing coagulation of its contents by the injection of some coagulating fluid like a solution of tannin or of perchloride of iron.

In malignant pulsating tumors of the orbit, complete extirpation of the mass is the only method which promises any satisfactory results.

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TUMORS OF THE ORBIT.

In considering the subject of tumors involving the orbit, it is well to distinguish between those neoplasms which originate in the orbit, either in the orbital tissue itself, the sheath of the optic nerve, or the periosteum of the bony walls, and those which arise in some one of the neighboring bony cavities and which involve the orbit secondarily. This distinction is important, not only from the diagnostic stand-point, but also from the side of surgical interference; for the location and extent of a growth involving the orbit frequently decide not only the question of an operation, but also the nature and extent of the operation itself. Many of the so-called orbital tumors arise in the ethmoid cells, the sphenoid antrum, the frontal sinus, the naso-pharynx, or the maxillary antrum. It is a well-known fact that tumors of the bones of the skull, or of the sinuses contained within these bones, may, and generally do, extend in every direction from one sinus or labyrinth to another, and often exist for a long time and reach a large size before appearing in the orbit. The growth of these tumors is generally slow and insidious, though occasionally it is exceedingly rapid. A very extensive disintegration of the ethmoid, sphenoid, and superior maxillary bones from tumors starting in their respective sinuses may have already occurred before the presence of such a growth is manifested in the orbit by the usual signs of protrusion or displacement of the eyeball, pain on pressure along the orbital walls or directly backward, and the visible or tangible presence of the growth itself within the orbit.

These tumors, in the literal narrow sense of swellings, may be either fluid or solid. The former almost always arise in the frontal sinus or ethmoid cells, and contain pus or mucus. The latter are either solid or densely gelatinous, are always malignant in character, and arise in the maxillary, sphenoid, or ethmoid antrum. Finally, a by no means uncommon growth in this locality is a bony tumor or a real ivory exostosis, arising from the bony walls surrounding the orbit.

TUMORS ORIGINATING IN THE ORBIT.

Under this head we include not only solid neoplasms, but also cysts of all kinds and sanguineous tumors classed under the collective name of orbital aneurisms. Tumors of the orbit are relatively frequent, forming nearly fifty per cent. of all orbital diseases, and Billroth has reported eighteen cases of orbital tumor occurring in a total of two hundred and seventeen cases of tumors of the region of the face. In children, tumors of the orbit occur next in order of frequency after tumors of the eyeball.

Symptoms and Diagnosis.—The most marked symptom of orbital tumor, which is absent only in the rarest cases, is exophthalmos. The degree of protrusion of the eyeball enables us to draw some conclusions as to the

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extent of the growth. Still, tumors which arise at the extreme apex of the orbit are slower in causing protrusion of the eye than those which originate farther forward, and the exophthalmos is less marked. The direction in which the protrusion occurs is of importance in determining the location of the tumor, it being usually on the opposite side of the orbit from the protrusion. The exophthalmos is occasionally accompanied by rotation of the eyeball.

Every displacement of the eye due to an orbital growth is accompanied by more or less marked limitation of its motility. Some authors recognize two varieties of this loss of motility, one mechanical and the other functional, the latter being due to a diminution of muscular power or activity. The mechanical variety corresponds to the location and size of the tumor, and is of diagnostic importance in locating the tumor, especially when the loss of motility is in only one direction. The eyeball remains immovable in this one direction, and, if the vision is unimpaired, diplopia results. The functional loss of motility is due partly to stretching or twisting of the muscles, as in high degrees of exophthalmos, and partly to disease of the muscle from extension of the neoplasm into the muscular tissue, or from atrophy due to pressure. In some cases the immobility of the eyeball is due to paralysis of the motor nerves, and here the origin of the tumor has probably been in the vicinity of the superior orbital fissure.

Another symptom which accompanies tumors of the upper part of the orbit is *ptosis.*

Digital examination will sometimes aid us materially in forming an opinion as to the situation, extent, shape, resistance, and movability of the tumor, assisted by puncture with needle or trocar, or by removing a small piece of the supposed tumor by the harpoon or knife and forceps for examination. Disturbances in the sensory nerves and anomalies of circulation, both circumscribed and extensive, will aid us in diagnosis. Spontaneous pain and sensitiveness on pressure are frequently, though by no means always, present. The spontaneous pain is generally constant and very wearing in character, but occasionally it is intermittent and sharp, like ciliary neuralgia.

Another symptom which is occasionally met with is pulsation of the eyeball or orbital contents, which may be felt or heard and sometimes seen, and is described by the patient as a "whizzing." It occurs in angiomata, in encephalocele, in angio-sarcomata, and in all forms of orbital aneurism. The tumor is sometimes increased in size by venous stasis, but this is more often than otherwise a seeming increase due to a positive increase in the intra-orbital pressure.

The disturbances of vision produced by an orbital tumor are of much the same nature as those met with in orbital cellulitis, and vary in character and intensity. There may be an artificial hypermetropia by flattening of the eyeball from before backward by the pressure of the tumor, partial or complete paralysis of the muscle of accommodation and of the iris (mydri-

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asis) from pressure on the corresponding branches of the third nerve, loss of central vision, central scotomata, narrowing of the visual field, and more or less complete amaurosis from inflammation or paralysis of the optic nerve. At first the ophthalmoscopic examination is negative, but as the pressure from behind continues and the obstruction to the return circulation becomes more complete, the retinal veins become engorged, the arteries narrowed, and the picture resembles finally that of papillitis, with hemorrhages in the retina and more or less extensive retinitis, ending in atrophy of the optic nerve. Occasionally we meet with primary atrophy of the optic nerve without any signs of neuritis. All these symptoms may be induced by the mechanical pressure of the neoplasm. In addition to the hemorrhages in the retina and on the disk, there may be thrombosis of the central retinal vein.

Sometimes there is a distinct increase of the intra-ocular pressure. In some cases a neuro-paralytic keratitis develops, with ulceration, abscess, and perforation of the cornea, due to interference with the nutrition of the cornea by the pressure in the orbit.

A comparatively rare complication is the extension of the orbital growth to the tissues of the eyeball, and consequent loss of the eye.

The chief danger to life in cases of orbital tumor is found in the extension of the growth into the anterior or middle fossæ of the skull. The tumor may extend backward through the optic foramen or superior orbital fissure to the middle fossa, or it may penetrate into the anterior fossa by caries and absorption of the roof of the orbit. More rarely a fatal result is brought about by the development of metastatic growths within the cavity of the skull.

As the tumor grows, all the contents of the orbit are pushed before it, until in aggravated cases the eyeball is protruded almost entirely outside the lids, the eyelids becoming enormously distended and congested, the lacrymal gland is displaced or atrophied, and the growth begins to extend outside of the limits of the orbit, upon the forehead, outward upon the temple, and downward upon the cheek.

The tendency of strictly orbital tumors to extend forward and outward, rather than backward towards the brain, is decidedly marked, and has been commented upon by many authors.

Topographically, orbital tumors may be divided into four classes:

1. Those which arise in the orbital cellular tissue, and which form the great bulk of all the tumors.
2. Tumors originating in the lacrymal gland.
3. Tumors originating in the optic nerve.
4. Tumors arising in the bony walls of the orbit.

It is by no means uncommon for recurrent orbital tumors to involve the periosteum and bone, even when all possible precautions have been taken against their return at the time of their removal by operation. In many of these cases some of the morbid cellular elements have been left in the

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shreds of orbital tissue, or in the many fissures and sinuses communicating with the cavity of the orbit. It is not improbable, however, that in some cases the periosteum or bone may have been the primary seat of the disease, which in the majority of instances is either sarcoma or myxo-sarcoma, and more rarely fibro-sarcoma. When a morbid growth in the orbit has been removed, the chances of its return are much smaller if it has been found encapsulated; and when in such a case the orbit again becomes filled, we must assume either a nidus of disease left behind or a new secondary tumor.

The secondary processes observed in bone and periosteum are a general infiltration, softening, and degeneration of bone-tissue, and the development of exostoses and osteophytes from and in the walls of the orbit. This process of infiltration is strictly one of disintegration, in which the apparent destructive power of the giant cells seems to be paramount. But in this pathological infiltration of the bone with sarcomatous elements there is little or no intercellular substance. When the osteophytes are met with in the orbit, they show a special predilection for the roof and inner wall of the orbit, being always attached to the periosteum or to the bone itself. Their development, as the result of the metamorphosis of the cellular elements of plastic inflammation, seems to be not a very uncommon complication of recurrent orbital sarcomata. In some instances this process of bone-growth may be seen by the side of bone-disintegration, produced by the infiltration of the bone-tissue with the myeloplaxes of sarcoma. How far these processes go on side by side, how intimate the connection between the two may be, and which comes first in order of time, are still unsettled questions, and some authors regard the two as different stages of the same process.

The influence of locality upon the development of sarcomata is said to be as evident in the orbit as elsewhere in the body, the osteo-sarcomata appearing in the periosteum and on the surface of the bones, while the softer forms, rich in cells, arise in the orbital tissue. A myxomatous element is very often present in sarcomatous tumors of the orbit, especially in the recurrent growths. As a rule, the more developed the cellular element of a sarcoma is the more rapidly does it grow and the farther it extends, and this is especially true of orbital sarcomata. Here the neoplasm does not grow in one direction only, but in all directions along the small blood-vessels and lymphatics. When these tumors recur after extirpation, the secondary growths are always richer in cells than the original tumor, grow much more rapidly, and are more apt to be myxo-sarcomatous than purely sarcomatous.

The size and rapidity of growth of a tumor in the orbit are usually indicative of its nature, and its growth here seems generally to be entirely beyond the laws which govern ordinary development in the body. The great danger in malignant tumors is, of course, the extension of the growth into the cavity of the skull. This may take place through the normal anatomical canals or openings, such as the optic foramen or sphenoidal fissure, or by destruction of the roof of the orbit by caries. In operating for the removal of a primary intra-orbital sarcoma, careful search should

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be made through the entire orbit for the presence of enlarged lymphatics or infiltrated glands, as these are a common channel of propagation and a frequent cause of the recurrence of the tumor.

TUMORS WHICH ARISE IN THE CELLULAR TISSUE OF THE ORBIT.

I. **Cysts.**—Under the head of cystoid tumors may be classed *encephalocele* of the orbit, which may be defined as a hernia from the brain, the sac of which is formed by the dura mater. The hernial ring on the intracranial side is, as a rule, formed by a hole or opening in the suture between the ethmoid and frontal bones, at the expense of the horizontal plate of the ethmoid. On the side of the orbit the exit is found most frequently between the frontal bone, the nasal process of the superior maxillary, and the lacrymal bone, and the latter is sometimes entirely absent. The skin covering the tumor is, as a rule, normal, though sometimes firmly adherent to the sac. An encephalocele is always congenital, and is due to an arrest of development in the bony walls. It is most frequently met with in the region of the lacrymal bone, and is often bilateral. The amount of displacement of the eyeball depends upon the position of the hernial ring. Sometimes all signs of a connection between the tumor and the cavity of the skull are absent: there is no pulsation, and pressure upon the tumor neither diminishes its size nor causes cerebral symptoms, and in these cases the diagnosis is rendered very difficult. An encephalocele is sometimes accompanied by an angioma, and this complicates the diagnosis, for pulsation may exist, the tumor may be compressible, and may increase in size during forced expiration. As a rule, an encephalocele increases rapidly in size, and most of the little patients die within the first few weeks or months after birth, either in consequence of the original hydrocephalic process or of diffuse meningitis developed from the gangrenous inflammation of the very tense skin overlying the sac. These cases do not admit of any treatment, either medical or surgical.

Pure Cysts.—There are several varieties of pure cysts developed in the orbit, which, according to their pathogenetic character, may be divided into extravasation cysts, pigment cysts, exudation cysts, retention cysts, and dermoid cysts. The latter class is a very large one, and has been subdivided into hygromata, oily cysts, fatty cysts, atheromata, steatomata, etc. All these are the results of defective development, and are congenital. The exudation cysts and retention cysts, so frequently described, all belong either to the general class of dermoid cysts or to the echinococcus cysts.

Extravasation Cysts.—Under this head may be included the blood cysts, hæmatocele, and hæmatoma, which present more or less characteristic cystic formation with bloody contents. This class does not include the free hemorrhages into the retro-bulbar connective tissue, which have been described under the head of "Hemorrhages into the Orbit." Almost all the cases of cystic formation in the orbit with bloody contents have been originally

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cases of dermoid cysts, in which exploratory puncture for diagnostic purposes has produced a hemorrhage, or in which the bloody contents have been caused by some other form of traumatism. Many of these dermoid cysts are very vascular, and the vessels lying on the inner wall of the cyst might very easily rupture during the mere process of growth. There are only two or three cases, at the most, on record which may be regarded as true blood cysts of the orbital tissue, with a cyst-wall and fluid and solid blood-contents, which have originated in hemorrhagic extravasations. The so-called "pigment cysts," or melanotic cysts, may be included in this class of "extravasation cysts," as they are of very doubtful origin, and are probably blood cysts which have undergone some pathological metamorphosis.

Exudation Cysts.—If we leave out of consideration the subject of dropsy of the capsule of Tenon, which histologically is not strictly a cyst, there is but a single variety of exudation cyst, which has been described as the *hygromatous degeneration of the orbital bursa.* The tendon of the superior oblique muscle is surrounded in the trochlea by a bursa, and there are similar bursæ between the tendons of the levator palpebræ and superior rectus muscles. Both Hyrtl and Demarquay assume that orbital hygromata arise in these bursæ, and other authors hold that all serous cysts of the orbit have their origin in the same bursæ. These cysts contain a serous or synovia-like fluid.

Retention cysts form quite a large class by themselves, according to De Wecker, who calls them "follicular cysts." He includes under this head the atheromata, steatomata, and cholesteatomata, and asserts that they all originate in the skin-follicle, because they are more or less closely connected with the eyelids. For this positive assertion there are no absolute data, and it is the generally accepted view among pathologists that all follicular cysts are simply varieties of true dermoid cysts.

Dermoid Cysts.—Dermoid cysts are to be regarded as fœtal structures resulting from the invagination or involution of the external blastodermic membrane. They are by no means rare occurrences in the orbit. They form a group of cysts by themselves under the name of teratoma. They are round and usually unilocular, though they may be multilocular, and their walls are quite thick, very vascular, and generally lined with epithelium. They are situated in the orbit outside of the limits of the funnel formed by the ocular muscles, and they probably arise in the anterior portion of the orbital cavity. They are more frequently found on the inner side of the orbit than eleswhere. They increase slowly in size and may reach an extraordinary development, but are of a benign character. They may be closely connected with the muscles, or the eyeball, or the sheath of the optic nerve, or the periosteum. Their contents may consist of epithelium or epidermis, hairs, fluid fat and fat crystals, fluid or gelatinous or solid constituents, and even skin and teeth, with occasionally chalky deposits. Microscopically, the fluid or gelatinous contents of these cysts cannot, probably, be differentiated from those of true atheroma.

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Congenital Cysts of the Orbit with Microphthalmos.—This is a peculiar congenital malformation, consisting in the presence of a large, transparent vesicle situated between the skin and the conjunctiva of the lower lid, which pushes the lid forward and sometimes extends deeply into the orbit. It is always associated with microphthalmos or anophthalmos. Various views have been held in regard to the true nature of these cysts, that of Berlin being probably the most correct. Berlin believes that the cystoid structure has proceeded from those embryonic structures from which a normal eye is usually developed. In the majority of the reported cases the cyst was unilateral, though it has been found in both orbits.

Echinococci of the Orbit.—Echinococcus cysts are occasionally met with in the orbit, the cyst being surrounded by a framework of connective tissue. The cavity is filled by the so-called mother cyst which is developed from the original embryo. This mother cyst contains the cysts of the third generation floating in a liquid which gives it its fluctuating character. They are very rare in the orbit, but have been found even within the lacrymal gland and in the frontal sinus. They have been known to extend from the orbit into the cavity of the skull, by gradual absorption of the roof of the orbit. A frequent—almost constant—symptom is ciliary neuralgia. As these cysts increase in size they may produce most of the symptoms which accompany any increase in the intra-orbital pressure. The fluid contents of such a cyst should be carefully examined to determine the presence of the small cysts, which are of great diagnostic importance.

Cysticercus in the Orbit.—This is a rare occurrence in the tissue of the orbit, though by no means an uncommon occurrence in the eyeball, especially in North Germany. In all the reported cases the cysticercus was found in the anterior portion of the orbit. The sac is usually surrounded by a very thick envelope of connective tissue. The symptoms are fluctuation of the tumor, redness and sensitiveness of the skin of the lids, and in some cases pain. If the cyst is of considerable size, the eyeball is displaced.

All these cystoid tumors in the orbit are characterized by fluctuation. The encephaloceles are very rare, and are distinguished by their congenital origin, their location, their bilateral character, and their pulsation. In doubtful cases an exploratory puncture would probably aid in the diagnosis. The question of differential diagnosis becomes important in the cases of dermoid cysts and echinococci, as the same symptoms exist in both, with the exception of congenital origin, which does not occur in the latter. In the case of an echinococcus ciliary neuralgia is usually present, while it is absent in a case of dermoid cyst. Berlin refers to a peculiar whizzing sound reported by Piorry to have been heard in cases of echinococcus, which the latter regarded as characteristic of hydatids. Exploratory puncture and microscopic and chemical examination of the contained fluid will probably aid us in making a diagnosis, though in the case of these two varieties of cyst the diagnosis will have no effect upon the prognosis or treatment.

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Treatment.—The treatment of all these cystoid tumors should be surgical, and the operation should be the total excision of the cyst. Some authors have recommended puncture or incision with subsequent cauterization of the sac or the introduction of a seton, and still others have advised partial excision. The disadvantages of all these methods, except the total extirpation, lie in the subsequent adhesions between the orbital tissue and the eyeball and the consequent limitation of motility. Hence under all circumstances the only operation that promises a satisfactory result is that of total extirpation of the cyst, under the most complete antiseptic precautions.

II. **Angiomata of the Orbit.**—Angiomata of the orbit are of three varieties,—simple angioma, cavernous angioma, and lymphangioma.

Simple angioma includes the nævus maternus and the telangiectasiæ of the lids and orbit, whether congenital or not.

Cavernous angioma includes the aneurisms by anastomosis and the erectile tumors. The characteristic symptom of the latter class is the protrusion of the eyeball in consequence of venous stasis.

Simple angioma is rarely met with in the orbit alone, for it is usually associated with angioma of the eyelids or neighboring skin. When there exists a simultaneous hypertrophy of the adipose tissue, the tumor receives the name of angioma lipomatodes. Cavernous angiomata are met with in all parts of the orbit, but are more frequently found behind the eyeball, within the cone formed by the ocular muscles, and are very often surrounded by a thick capsule of connective tissue.

Symptoms and Diagnosis.—Simple angiomata are usually associated with a similar condition of the eyelids and neighboring skin, and appear as soft, slightly compressible, tumor-like prolongations of the telangiectasia into the orbit. They are almost always congenital. Cavernous angiomata, if superficial, give a bluish appearance to the subcutaneous tissue. They present spontaneous pulsation, are firmly elastic but not dense, of slow development, and usually painless. There is not much interference with the motility of the eyeball, especially if the tumor is situated within the funnel of the muscles. The compressibility is somewhat variable, and it should not be forgotten that other varieties of orbital tumor are also compressible. Pulsation, though often imagined, has never been made out with any certainty.

Lymphangioma is excessively rare, and its symptoms differ in no way from those of cavernous angioma.

Course and Prognosis.—The course is extremely slow and gradual in the cavernous form, but in the simple and telangiectatic forms it is much more rapid. The prognosis is favorable, for hemorrhages are excessively rare, and some cases actually heal spontaneously. The chief danger lies in progressive loss of vision from pressure on the optic nerve, though these tumors have been known to destroy the eye by progressive inflammation.

Treatment.—The treatment should always be operative, and in the

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majority of cases, particularly of the cavernous variety, total extirpation of the tumor gives the best results. In some cases of congenital telangiectasia of the lids and orbit, electrolysis has succeeded in producing a gradual diminution in the size of the tumor and a gradual improvement in the condition of the lids. The operation must here be repeated a number of times at comparatively short intervals, in hopes not only of producing clots in the enlarged vessels and cavities, but also of inciting an adhesive inflammation of the walls of these blood-vessels and cavities, and thus gradually obliterating the tumor.

III. **Lipoma of the Orbit.**—True lipoma of the orbit is a rare disease, and must be differentiated from a general hypertrophy of the fatty connective tissue of the orbit which is occasionally met with in Basedow's disease and certain other general pathological conditions. Fatty tumors of the subconjunctival connective tissue and of the lids occasionally extend into the orbit, and in a sense may be regarded as lipomata of the orbit, but there are very few cases on record of a lipoma actually originating in the fatty tissue of the orbit, and even these are somewhat questionable. The tumor reported by Knapp as an angioma lipomatodes, and already referred to, was a case of secondary hypertrophy of the adipose tissue around an angioma, and therefore cannot strictly be placed in this category.

IV. **Enchondroma of the Orbit.**—The cases hitherto reported of enchondroma of the orbit are described as tumors of a cartilaginous character developed in the fatty tissue of the orbit or in the lacrymal gland. Berlin is inclined to classify them all with the cylindromata. Even the case reported by Fano is obscurely described as an osteo-fibro-cartilaginous tumor existing from infancy at the inner angle of the upper lid, and was hard and movable. It was encapsulated, was removed with the greatest ease, and found to contain cartilage-cells embedded in a dense fibrous tissue. Nothing is said about its possible connection with the eyelid, but the probability, at least, is that it had its starting-point in the tarsus.

V. **Orbital Tumors of Epithelial Character.**—Under this general classification various authors have arranged three different varieties of tumor having certain general resemblances,—the epithelial carcinoma, the adeno-carcinoma, and the adenoma. Strictly speaking, none of these tumors arise in the orbit, but all start from the lids, or from the lacrymal gland, or from the eyeball, or in some one of the neighboring cavities, and extend into the orbital tissue secondarily. Such growths will therefore receive their proper consideration elsewhere.

VI. **Orbital Tumors of the Connective-Tissue Type.**—Under this head are included *sarcoma*, both the round-cell and the spindle-cell varieties, *fibro-sarcoma*, *myxo-sarcoma*, *cysto-sarcoma*, *cylindroma*, and *plexiform sarcoma*. Most of these orbital growths present about the same symptoms. They are solid, with more or less uneven surface; there is neither fluctuation nor pulsation; they are neither compressible nor very hard; they have no direct connection, as a rule, with the brain, nor the lids, nor the eyeball;

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they may involve one or more of the neighboring cavities, but they are much more likely to originate in some one of these cavities and extend to the orbit secondarily. Still, if they are very vascular, they may pulsate and be compressible, and in the case of a myxo-sarcoma or a cysto-sarcoma there may be fluctuation. As regards the true nature of such a tumor, the microscope alone can establish the diagnosis, but in some cases the age of the patient, the rapidity of growth, the pain, the loss of motility of the eyeball, and the resistance of the tumor may all aid in revealing its real nature.

Cylindroma.—Modern pathology has practically decided that a cylindroma belongs to the class of sarcomata. It is marked by severe pain and a decided tendency to rapid recurrence. It has a typical alveolar structure and contains a relatively large amount of hyaline tissue. The cells which constitute the bulk of the tumor come from the middle blastodermic membrane, and the tumor is therefore of the connective-tissue type, whether the cells originate in white blood-corpuscles, or in connective-tissue corpuscles, or in the perivascular lymph-spaces, or in the cells of the adventitia of small blood-vessels. On the other hand, the alveolar structure and the tendency to rapid recurrence show a strong resemblance to the carcinomata, and for this reason Sattler has proposed the name of carcinomatous sarcoma in place of cylindroma. The prognosis is most unfavorable, and death results from extension of the growth into the neighboring cavities, including that of the skull, the intervening bony wall being worn away and absorbed. The treatment consists in an early and most complete extirpation of the tumor and the entire contents of the orbit.

The *plexiform sarcoma* resembles very strongly either a cylindroma or a myxo-sarcoma, and it seems unnecessary to make a separate classification for this growth. It is very rare.

Myxo-Sarcoma.—This is a by no means infrequent occurrence in the orbit. The symptoms are not characteristic of the nature of the growth, though a very rapid growth has been frequently mentioned in the cases reported. Under the microscope, such a tumor shows numerous fusiform and stellate cells contained in a fluid consisting largely of mucin.

Sarcoma.—This term includes the round-cell, the spindle-cell, and the fibro-sarcoma varieties, and this class forms the great bulk of all the orbital tumors that have been reported. It is impossible to distinguish these three varieties from each other, and even under the microscope the same tumor will be found to contain not only all the various cell forms, but even a pure fibro-sarcomatous structure. Some of these growths undoubtedly arise in the fatty tissue of the orbit, others in the fibrous capsule of Tenon, and still others in the episcleral connective tissue. Many of them have started from the periosteum lining the orbit, and not a few in the lacrymal gland. In some rare instances they have appeared as metastatic growths, the original tumor having started in some other part of the skull or body. The most frequent variety is probably the small round-cell sarcoma, in

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which, however, we meet with larger cells and even giant cells. Pure spindle-cell sarcomata are also common, and fibro-sarcomata are not infrequent. In this latter Berlin includes the fibromata. The so-called encephaloid tumors must be regarded as round-cell sarcomata. A cysto-sarcoma is simply a round-cell or spindle-cell sarcoma containing numerous hollow spaces filled with fluid.

The nature of a recurrent tumor cannot be predicated from an examination of the original growth, for the intimate structure of these tumors is so complex and variable that a round-cell or spindle-cell sarcoma frequently becomes a myxo-sarcoma or glio-sarcoma without masking any of the usual symptoms. The course and results of all sarcomata are very variable. The younger the individual is and the more numerous the cellular structures are, the more rapid is their growth and the greater certainty is there of a return.

The treatment consists in complete extirpation at the earliest possible period.

Melanosarcoma.—In the great majority of cases of orbital melanosarcoma the tumor is not primarily an orbital growth, but extends to the orbit from the choroid, or from the subconjunctival tissue near the margin of the cornea, or from the cavity of the skull. Occasionally, however, it has been found as a primary growth in the orbit, and it has been known to perforate the sclera from without and enter the eyeball. In some of the cases reported the tumor originated in the periosteum. The histological nature of these tumors is still but little understood. They have been described as pure melanosis of the orbital tissue, but it is sufficient to say that microscopically they consist of a mass of small round or fusiform cells arranged in more or less complete alveoli, and containing a strongly marked pigmentary deposit not only in the cells, but scattered all through the connective-tissue framework.

Before operation the diagnosis must largely depend upon the dark color of the tumor, and even then it might be confounded with a cavernous angioma.

The prognosis is always unfavorable, as in the case of other melanotic tumors. In spite of early and complete extirpation, the tumor is almost certain to recur, and in a very short period of time. It shows a strong tendency to extend towards the intra-cranial cavity and to the development of metastatic growths in various parts of the body.

Plexiform Neuroma of the Orbit.—This is a very rare growth, of which only a few cases have been reported, notably by Billroth, Marchand, and Bruns. It appears to be either congenital itself, or a subsequent extension of a congenital, extra-orbital neuro-fibroma. It has been found in the upper and outer angle of the orbit, near the lacrymal gland, and the eyeball is displaced forward, downward, and inward. It grows very slowly, causes no pain, and is not sensitive to pressure. The diagnosis is possible only when a similar growth exists outside of the orbit, which can be

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recognized. The prognosis is entirely favorable. The treatment consists in complete extirpation, and the tumor shows no tendency to return.

The prognosis of all forms of malignant orbital tumors, whether primary or secondary, is unfavorable; and if the tumor be primarily one of the deep facial bones or their sinuses, the prognosis is positively bad.

Except in the case of encapsulated tumors of the orbit, surgical interference is almost invariably followed by a return of the tumor, and the growth of the secondary tumor is more rapid than that of the primary lesion. With each succeeding operation the period of quiescence before the return of the tumor grows shorter and the rapidity of the growth increases.

Repeated operations in these cases undoubtedly shorten the life of the patient. While it is, therefore, our duty to operate in all cases in order to relieve severe or unbearable pain, we should be slow to operate merely for the sake of relieving temporarily physical disfigurement, especially if we are convinced that by so doing we shorten the life of the patient.

TUMORS WHICH ARISE FROM THE BONY WALLS OF THE ORBIT.

I. **Cysts of the Orbital Walls.**—The existence of such tumors is decidedly doubtful, in spite of the cases reported by various authors. Echinococcus and serous cysts of the orbital walls have been mentioned by Mackenzie and others, but they were probably cysts of the frontal sinus, which developed within the cavity of the sinus and not in the spongy portion of the frontal bone. Cholesteatomata have also been reported as arising from the interior of the frontal bone, but the origin of these tumors is equally uncertain.

II. **Osteo-Sarcoma.**—Almost the same criticism may be passed upon the so-called osteo-sarcoma of the orbital walls. In most of the cases reported the participation of the orbital walls in the process was only part of a very extensive disease of the other bones of the skull, and it was not possible to fix the origin of the diseased process in the bony walls of the orbit.

III. **Osteoma of the Orbit.**—Under the general head of osteoma are classed a number of bony growths of different internal construction and of varying external appearance. Osteophytes, periostoses, hyperostoses, and exostoses are all classified together for the sake of convenience, as outgrowths from the bony walls of the orbit, partly from the periosteum and partly from the diploë of the bones themselves. Some of these outgrowths are cellular in structure, a few are semi-cartilaginous, but the great majority are of ivory hardness, and it is to this latter class that the term osteoma has come to be restricted. These bony tumors are more frequently found on the upper and inner portion of the orbit than elsewhere, but, unless the attention of the surgeon is called to them early in their existence, it is rarely possible to determine accurately the exact point of origin of the tumor. The frontal bone seems to be the favorite seat of an osteoma, and next to the frontal bone comes the ethmoid. An osteoma may be unilateral or

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bilateral, and, though usually single, there may be several osteomata in the same orbit. These tumors are generally hemispheroidal in shape, with a broad base of attachment, and narrow pedicles are very rare. They vary greatly in size, and their surface is frequently uneven and even nodulated. Though commonly regarded as starting from the bony wall and projecting towards the orbit, they sometimes grow towards or into some one of the neighboring cavities, and in not a few cases the tumor has originated in a neighboring cavity and involved the orbit secondarily. Cases have been reported in which the tumor began in the ethmoid cells, in the nasal meatus, and in the frontal sinus. The latter are the most frequent of all, and by their pressure cause absorption of extensive portions of the bony wall, and eventually an opening into the anterior fossa of the skull. Such a course would render any prolonged attempt at removal of the tumor extremely hazardous.

Their *etiology* is extremely obscure, and it may be truthfully said that we know little or nothing about it. They have been attributed to constitutional syphilis, gout, rheumatism, scrofula, and traumatism; but, in view of our lack of accurate knowledge on the subject, it is as well to look for the predisposing cause, at least, in an abnormal embryonic condition of the bone cells.

Symptoms and Course.—Osteomata almost always grow very slowly and painlessly, and years usually elapse before they produce any severe symptoms. They are never sensitive on pressure, and when they cause pain it is either from pressure on some nerve of sensation or from some localized inflammation. As the tumor grows the eyeball is displaced, and there may be exophthalmos, occasionally to such an extreme degree that the eyelids cannot close over the eye, and the cornea ulcerates. If the exophthalmos is at all marked, there is usually marked disturbance of the vision. Ophthalmoscopically there may be noticed all the changes in the retina and optic nerve, from simple hyperæmia, through papillitis, to atrophy of the optic disk. If the tumor has encroached upon the cavity of the skull, grave cerebral symptoms may result, though generally this is not the case, owing to the extreme slowness of growth.

Diagnosis and Prognosis.—The diagnosis of an osteoma of the orbit is not usually difficult, though there are several lesions with which it may be confounded. Its extreme hardness, its immobility, and its direct connection with the bone, all point to a bony growth. If at the supero-internal angle of the orbit, it may be confounded with a distention of the frontal sinus. If along the inner wall of the orbit, it may be mistaken for a distention of the ethmoid cells. Hence, in making our diagnosis, a careful examination must be made of the condition of the various neighboring cavities, as these bony tumors frequently extend into these cavities and not infrequently start from them, both of which conditions complicate both the prognosis and the treatment. If, however, the tumor has not extended into the cavity of the skull, the prognosis is generally very favorable, and it is often possible

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to preserve the eyeball intact, though the vision may be seriously and permanently affected, and perhaps even entirely destroyed.

Treatment.—The treatment of pure osteoma of the orbit, or ivory exostosis, consists solely in its operative removal, and if it be of large size this must be accompanied by enucleation of the eyeball. Owing to the extreme hardness of the tumor, its removal is usually very tedious and a matter of considerable difficulty, and the danger to life of such an operation is by no means slight. If the tumor grow from the roof of the orbit, the danger is greater. In the fatal cases death results from meningitis or from a cerebral abscess in the vicinity of the seat of operation; and if the tumor be of considerable size here, it is wiser not to attempt its removal. In such case the eyeball may be enucleated in order to relieve the severe pain which is sometimes present. The extreme danger involved in attempting to remove osteomata arising from the roof of the orbit may be seen from statistics collected by Berlin. Out of a total of thirty-two cases of orbital osteomata, sixteen were attached by their base to the roof of the orbit, and of these six died,—a mortality of thirty-eight per cent.

In operating for the removal of osteomata, the periosteum must first be incised and carefully stripped off from the tumor and for some little distance from the base in all directions. Then a narrow groove should be cut with a chisel or gouge and mallet around the base, the blows with the mallet being made gently and somewhat rapidly until the tumor becomes loosened. Then a gentle rocking to and fro will usually suffice to detach it. If the tumor be found of ivory hardness, small holes may be drilled in it by means of a dental engine, and the operation subsequently completed by means of the chisel and mallet. If the periosteum is first carefully stripped off, the tumor may sometimes be shelled out by a very few strokes of the mallet, much more readily than would be at first supposed.

DISEASES OF THE CAVITIES ADJACENT TO AND SECONDARILY INVOLVING THE ORBIT.

The frontal sinus, the ethmoid cells, the sphenoidal sinus, and the maxillary antrum are often the seat of disease which subsequently extends to the orbit. A brief glance at the anatomical relationship of the bones forming these cavities, and at the base of the skull, will enable us to understand more clearly the nature of these diseases, and the important relation they bear to the cavity of the orbit.

DISEASES OF THE FRONTAL SINUS.

The frontal sinuses are situated within the frontal bone, at the level of the nasal boss of that bone, just above the root of the nose. They are usually two in number, separated by a more or less complete partition of bone, which is generally, though not always, in the median line. Their dimensions are very variable, but their size usually increases with the age

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of the patient. Their form is very irregular. The anterior wall is thickest, while the cranial and orbital walls consist mainly of compact bony tissue with a thin layer of cancellous tissue in the centre. Each frontal sinus opens into the infundibulum of the ethmoid by a canal in the anterior cells of this bone, called the frontal or fronto-nasal canal. Each sinus is lined by a thin, smooth, rosy mucous membrane, slightly adherent to the bone, and consisting of two layers, one mucous, the other periosteal. In young subjects the sinus does not exist, but after adult life is reached its size presents the most remarkable variations.

The most frequent diseases met with in the frontal sinus are: first, *mucocele*, or a collection of mucus; and, second, *empyema*, or *abscess*. Both of these are chronic diseases, and the symptoms are practically the same in both. Distention of the frontal sinus can generally be easily recognized by the site of the swelling. It is higher up and farther back than the site of the lacrymal sac, and it has hard, resisting walls. Occasionally a spontaneous opening occurs by absorption of the bony wall, and the symptoms are then those of a cyst.

Empyema, or *abscess* of the frontal sinus, is usually an affection of adult life, though it is met with most frequently between the ages of twenty-five and thirty. It does not occur in childhood, as the sinuses are not developed to any extent at this period of life. It is found oftener among men than among women, and this is probably due to anatomical peculiarities such as the following: the sinuses are much more developed in men than in women; the infundibulum is wider and communicates more directly with the nasal fossæ, and through the latter with the external air. Hence infectious agents may more easily enter them. The left frontal sinus is more frequently attacked than the right, and the cause of this is as yet unknown. It is not uncommon to find at the time of operation that the suppurative process began in one sinus, perforated the bony septum, and involved the other sinus secondarily.

The etiology of suppuration of the frontal sinuses is somewhat obscure. It has been attributed to tuberculosis and syphilis, but this is not strictly correct. Both these constitutional affections may produce an osteo-periostitis of the frontal bone, accompanied or followed by extensive caries or necrosis and profuse suppuration, and the frontal sinus may be invaded by the pus, but always in the direction from without inward. The same argument applies to erysipelas, though here it must be admitted that the diseased process might extend through the mucous membrane of the nasal fossæ to the infundibulum, and thence to the frontal sinus. The sinuses have also been known to suppurate during the decline and convalescence of scarlatina and typhoid fever and during a severe attack of influenza. Wounds of the superior orbital margin may possibly produce abscess of the sinuses, but only after a long lapse of time, the diseased process extending gradually through the bone. Foreign bodies, whether *inert* or *alive*, on entering the nasal meatus and penetrating to the sinus may set up a

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chronic suppurative process. Inflammatory lesions of the mucous membrane of the nasal fossæ are found to exist in many cases of abscess of the frontal sinus. Finally, polypi of the nasal fossæ have been known to precede the development of abscess of the sinus.

All these are of course mainly predisposing causes. In order that suppuration should occur in a frontal sinus, there must be an entrance and proliferation of pyogenic microbes in the cavity of the sinus. The latter may penetrate directly into the sinus without infecting the nasal fossæ, or the suppuration of the sinus may be induced by that of the nasal fossæ. In the latter case infection of the sinus may occur through the frontal canal or infundibulum, or by way of the lymphatics. Once invaded by the microbes, the mucous membrane of the sinus swells and becomes thicker, and thus the canal of the infundibulum is narrowed and eventually obliterated. Then the pus begins to accumulate in the sinus. In simple catarrh of the sinus the infundibulum becomes closed, but here the result is an accumulation of mucus and not of pus. If this is to become an abscess, there must be a second element of an infectious nature present. When a sufficiently large opening has been made into the sinus, and the pus has been discharged from the abscess, the mucous membrane lining the sinus is seen to be red and inflamed, swollen and thickened, sometimes to ten or twelve times its natural thickness. Its surface is rough, granular, and even fungoid. Large quantities of these fungoid excrescences may be removed with a curette or destroyed by the injection of some caustic. The disease may perforate the bony wall of the sinus naturally, usually the inferior wall, but always by a small opening; or it may perforate the cranial wall and enter the cavity of the skull. Here the dura mater usually limits the extent of the abscess. Sequestra of bone are not uncommon, but they are generally very small and float freely in the pus. When these cases yield rapidly to treatment they are of two kinds: either the abscess has been a recent one, which has produced superficial lesions of the mucous membrane, or there has been extensive necrosis. In the latter case the frontal and orbital walls of the sinus have been cast off, the cavity of the sinus has disappeared, and cicatrization has followed, leaving a deep pit or hollow in this region. The diagnosis is difficult, especially in the beginning.

Guillemain thinks there is only one constant symptom, the supra-orbital pain, which is sometimes intense. But there are many cases of abscess of the frontal sinus in which there is never any pain at any time during the development of the abscess. The disease usually resists all medication, and disappears only when the pus is evacuated. If with the pain there is coryza, ozæna, or a purulent discharge from one nostril, and the frontal boss is painful and protuberant, and if the eye is displaced downward and outward, the lesion is almost certainly in the sinus, though it may not be abscess; hence in these cases a thorough anterior and posterior rhinoscopic examination should always be made. At a more advanced stage of the

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malady, when the orbit is involved, the diagnosis may be still more difficult, unless the disease is of the acute variety, when some of the signs of acute inflammation may be present. Still, even here, while there may be displacement of the eye downward and outward, there is rarely any protrusion of the eye forward. A small orbital abscess lying behind the tendon of the orbicularis muscle, compressing the canaliculus and causing epiphora, might be confounded with abscess of the lacrymal sac, but if a probe can be passed into the sac the latter possibility is excluded. Some assistance in diagnosis is occasionally gained by what is called "transillumination" of the frontal sinus by means of the incandescent electric light. The best lamp for this purpose is probably that recommended by Dr. G. W. Caldwell, who prefers a large oval lamp of four or five candle-power, provided with a switch handle. This is run by a battery capable of developing from five to ten volts, and provided with a rheostat for regulating the current to the resistance of the lamp. For the examination of the frontal sinuses, the lamp, protected by soft-rubber tubing, is placed against the under surface of the supra-orbital ridge, just internal to the supra-orbital foramen. In health, the illuminated area corresponds to the extent of the frontal sinus, extending somewhat beyond the median line. If there is suppuration in the sinus, this area is in shadow, due to the pus or thickened mucous membrane.

Treatment.—If we are sure that an abscess of the frontal sinus is present, the indications are to open it as soon as possible, drain it carefully, and inject antiseptic solutions, and thus put a stop to the suppurative process. If it is probable that no extensive bone-disease is present, the sinus may be freely opened internally by enlarging the infundibulum through the nose with a curette, and thoroughly irrigated and medicated by means of a flexible catheter. If, however, it is probable that extensive necrosis is present, an external incision should be made, starting from near the inner canthus immediately beneath the superior orbital arch, and running directly outward from an inch and a half to two inches, so that if the bony wall of the sinus, which is here very thin, has not already been perforated, it may be easily opened. The pus then flows out, and the sinus should be thoroughly washed out with a solution of mercuric bichloride (1 to 1000). The opening should be large enough to admit the little finger, and the cavity of the sinus should be carefully examined for fungoid granulations and osteophytes, and these, if found, should be thoroughly removed. Then the septum between the sinuses should be examined for a perforation, for in some cases this partition wall has been found not only perforated, but nearly entirely absorbed. A drainage-tube should then be inserted, and the cavity carefully washed out twice daily. The re-establishment of the channel of communication between the sinus and the nasal fossa is generally advisable. This may be done by opening into the ethmoid cells by trephine or chisel and mallet, or by introducing a catheter through the fronto-ethmoidal canal, and then a drainage-tube through the nose, after

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the plan advocated by Panas. After the pus ceases to be discharged from the sinus through the drainage-tube the cavity generally begins to granulate from the bottom, and in from three to four months has usually entirely filled up and closed over.

Summary of Symptoms.—In chronic inflammatory disease of the frontal sinus there may or may not be supra-orbital pain. If the process is confined to the frontal sinus, there is no other symptom until late in the disease. If there is sensitiveness on pressure over the frontal boss, swelling along the lower surface of the supra-orbital margin and inner wall of the orbit, and displacement of the eyeball downward and outward, it is probable that the disease has extended to the ethmoid cells. If there are coryza, ozæna, and a purulent discharge from the nostril, the nasal meatus has become involved and the diagnosis is certain. If the first symptom of orbital complication is the appearance of a dense, hard swelling at the upper and inner angle of the orbit, along the superior orbital margin and region of the lacrymal bone, and if the growth is slow and painless, the disease is almost certainly an osteoma of the frontal bone.

DISEASES OF THE ETHMOID CELLS.

The air-cells or spaces of the ethmoid form a pneumatic labyrinth, which increases in width from above downward. From the uncinate process to the cavity of the skull the ethmoid is pushed into the cavity of the nose. Anteriorly it is connected with the lacrymal bone, and posteriorly it is often united with the orbital portion of the palate bone. The posterior ethmoidal cells and the cavity of the sphenoid bone open into the upper nasal meatus. In rare cases the ostium maxillare is absent, and the maxillary antrum communicates with the ethmoid cells and sphenoid antrum. Sometimes the ethmoid labyrinth sends processes into the frontal sinuses, and occasionally the lateral ethmoid cells project very markedly towards the orbit.

The *symptomatology* of the diseases of the ethmoid labyrinth is always perplexing, and a diagnosis is thus rendered extremely difficult. In inflammation of the ethmoid cells positive subjective symptoms are wanting. Inflammation of their lining mucous membrane can by no means be readily distinguished from inflammation of their bony walls. In *mucocele* of the ethmoid cells the symptoms are a gradual painless development of a tumor on the inner wall of the orbit, which later shows signs of fluctuation. The eye is pushed forward or outward and sometimes downward. Until fluctuation appears, it may be confounded with an osteoma growing from the inner wall of the orbit. Puncture or incision of the tumor will decide the diagnosis. It is very often connected with a similar condition of the frontal sinus, but it may proceed directly from the nasal meatus.

In intense suppurative inflammation the periosteum suffers also, and this may lead to necrosis. It is probable that in the many cases of nasal disease in which orbital abscess is observed, the inflammatory process ex-

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tended from the nose to the ethmoid cells, and thence to the orbital tissue. The reverse may also be the case, the disease starting in the orbit and extending to the ethmoid cells, and thence to the nose. Collections of pus in the frontal sinus may lead to orbital abscess, and even to abscess of the brain, through the medium of the ethmoid cells. An inflammatory process may extend from the maxillary antrum to the ethmoid cells, orbit, and brain, the walls of the ethmoid cells here becoming absorbed by pressure from the abscess. In caries of the lamina papyracea the subjective symptoms are dull pain, increased by pressure in the neighborhood of the diseased bones, and vertigo. Redness of the lids at the inner canthus may be present. A hard tumor may be felt at the inner canthus, and later occur fluctuation, strabismus, diplopia, exophthalmos, and limitations of the movements of the eyeball, and the vision becomes reduced. The posterior ethmoidal cells open in the nose above and below the middle turbinated bone, and may be observed in the rhinoscopic mirror. Pus found in the region of the middle turbinated bone must come from these posterior ethmoid cells. Transillumination of these cells by means of the incandescent light gives no certain or satisfactory aid in determining their condition.

In all cases of inflammation, the opening of the ethmoid cells, evacuation of pus, and removal of fragments of carious bone are justifiable and necessary, and there may also be indications for opening some of the other adjacent cavities.

Tumors of the Ethmoid.—A morbid growth confined within the ethmoid cells gives rise either to no symptoms at all, or merely to headache paroxysmal in character. The orbital symptoms are the same as those of tumor of the orbit. The motility of the eyeball is limited. The vision may be slightly affected, or there may be complete blindness. The visual field may not be involved. If the tumor has entered the naso-pharynx, the mouth is more or less open and the speech is nasal. Later there is loss of the sense of smell. There may be more or less dropping of clear fluid from the nose, even in the case of solid tumors, owing to a communication between the upper wall or roof of the ethmoid cells and fissures at the base of the skull. There may also be orbital palpebral emphysema and hemorrhage from the nostrils on one or both sides.

Enchondroma of the ethmoid is very rare, and always starts from the base of the skull.

Polypi originating in the ethmoid cells and confined within these limits are relatively rare, but nasal polypi usually start from the ethmoid. Polypi of the naso-pharynx, on the other hand, not uncommonly penetrate the ethmoid cells. When the bridge of the nose seems widened, the orbit is narrowed by the pushing outward of the inner wall of the orbit, and protrusion of the eyeball in various directions is apt to result.

Fibroma originating in the ethmoid itself has been observed but once, and hence it is generally believed that a fibroma involving the ethmoid usually starts from the bones of the nose.

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Osteoma of the ethmoid bone generally begins in some neighboring cavity. The first objective symptom is a very hard tumor at the inner canthus of the eye. Then follow swelling of the neighboring part of the cheek, protrusion of the eyeball forward and outward, and diplopia. The inner canthus is pushed forward, as are also the nasal and lacrymal bones. The tumor also usually involves the nasal meatus, pushes the septum nasi to one side, and closes one or both sides of the meatus. It pushes the hard palate downward. The vision may be normal or impaired. There may be papillitis and suppuration of the cornea from an inability to close the lids completely over the eye. If the osteoma be encapsulated, it may easily be separated from its bony attachments. These osteomata never tend to penetrate the cranial cavity, and in this they differ from bony tumors of the frontal sinus. Their operative removal is not, as a rule, difficult. If, on the contrary, they arise from the frontal sinus and penetrate the ethmoid, their removal is usually a dangerous operation and gives bad results.

THE SPHENOIDAL SINUS OR ANTRUM.

The anterior, posterior, and lateral walls of the sphenoid antrum are always thin, and sometimes very thin. The anterior wall may be entirely wanting, and then the sphenoid sinus opens into the ethmoid cells. The hollow spaces in the ethmoid and sphenoid bones are by some regarded as a respiratory organ for the anterior and middle fossæ of the skull. However this may be, the anatomical relations between the ethmoid and the sphenoid are so intimate that any chronic process, such as a morbid growth, starting in the sinus or cells of either bone is almost certain to involve the other at a comparatively early date. The connection between the sphenoid antrum and the cavity of the nose is by openings in the posterior wall of the nose, at its extreme upper portion, opposite the superior turbinated bone. The subjective symptoms of abscess of the sphenoid antrum are the same as those of empyema of other neighboring cavities, though the headache is located in the back of the head, and the ocular lesions are either purely functional or affect the optic nerve back of the eyeball. Attempts have been made to treat the interior of the antrum through the nose, but the position of the opening into the nose is so variable, and the use of the rhinoscopic mirror is so unsatisfactory, that such attempts have usually been abandoned.

Disease of the body of the sphenoid, whether ending in caries and necrosis or not, may cause not only exophthalmos, but disturbance of vision, on account of the close proximity of the optic canal. Pain occurring in the course of disease of the body of the sphenoid may show itself in a totally different part of the area of influence of the trifacial nerve, and thus lead to a faulty diagnosis.

Tumors of the Sphenoid.—So long as a pathological process, whether it be inflammatory or a new growth, is limited to the sphenoidal antrum,

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either the subjective symptoms are entirely absent, or there may be severe pain in the head. If the process extends to the adjacent structures, symptoms arise which point to the probability that the sphenoid bone is the seat of the disease, such as blindness due to compression of one or both optic nerves, and the visible or tangible presence of the growth in the naso-pharynx, ethmoid, orbit, or skull. The entrance of the growth into the cranial cavity may occur without any subjective symptoms, or there may be severe headache. If the progress of the growth is very rapid, meningitis or cerebral abscess will result. The ophthalmoscopic symptoms are either papillitis or atrophy of the optic nerve due to perineuritis and pressure of the swollen nerve-sheath on the optic-nerve fibres. In some cases the pressure is exerted on the optic nerve in the optic canal. Tumors of the sphenoid antrum may perforate the middle fossa of the skull without causing blindness, and when loss of sight does occur in these cases it is not necessarily due to pressure on the optic chiasm, for it may be unilateral. If an orbital tumor rapidly causes blindness, and the latter starts from the temporal side of the field and leaves the region of the macula lutea unaffected to the last, and if at the same time a growth appears in the naso-pharynx, it is probable that the tumor began in the sphenoid antrum.

Polypi in the sphenoidal antrum may develop there independently, or they may originate in the naso-pharynx and penetrate thence into the sphenoidal sinus and ethmoid cells. They may also perforate the bone and enter the middle fossa of the skull, and even cause meningitis, without giving rise to any disturbance of vision.

Osteomata of the sphenoid may start from the periosteum or the diploë. In many cases they are developed from the embryonic remains of cartilage, and sometimes arise in the cavities themselves. They tend to penetrate the cavity of the skull, and, by compression of the optic nerve in the optic canals, early lead to blindness of both eyes.

Hyperostosis and *exostosis* of the sphenoid may produce the same orbital and ocular symptoms as osteoma of the sphenoid does.

Enchondroma of the sphenoid is excessively rare, only one case having been reported in literature.

Sarcoma.—In sarcoma of the base of the skull it is generally very difficult to determine the point of origin. Virchow says that sarcomata never start primarily from the mucous membrane of cavities, but from the underlying bone; and the mucous membrane is either secondarily affected or pushed forward by the growth. The general symptoms are here loss of sight, of hearing, and of smell, facial paralysis and neuralgia, vertigo, somnolence, vomiting, loss of memory, hemiparesis, and loss or impairment of speech. Death results from meningitis or encephalitis. There is no record in literature of sarcoma beginning primarily in the ethmoid or the sphenoid bone.

Carcinoma.—Only one case is reported where the carcinoma originated in the sphenoidal sinus.

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NASO-PHARYNX.

The intricate nature of the nasal meatus, with its many folds of mucous membrane, its spongy bones, its intimate connection with all the adjacent sinuses, and its proximity to the orbit, is amply sufficient cause for the extension of disease from this region to the orbit, and renders necessary a thorough examination into the condition of the meatus in all cases of suspected morbid growths.

Polypoid growths in the naso-pharynx may extend into the ethmoid cells and produce secondarily many of the symptoms of orbital disease. They may possess a partly cartilaginous consistency. It cannot be definitely determined whether all the visual defects which occur in polypi of the naso-pharynx are caused by pressure of the after-growth on the optic canal or in the ethmoid cells, and thence against the inner wall of the orbit. Whatever the cause, the loss of vision is due to pressure on the optic nerve.

Tumors of the Naso-Pharynx.—Tumors of the nasal and pterygo-palatine fossæ may enter the orbit through the infra-orbital fissure. They cause neuralgia of the infra-orbital or posterior alveolar nerves. The orbital portion of the tumor may divide into two branches, one involving the orbit and the other extending into the cranial cavity through the supra-orbital fissure. They eventually extend into all the neighboring cavities.

In the treatment of these growths it is absolutely necessary that they should be completely extirpated early in their development, together with all the surrounding tissues, including the bony walls of the cavities involved. If a malignant tumor has already invaded the deep bones of the face and base of the skull, including the cavities contained within them, the case may be regarded as hopeless, and while an operation may relieve the patient temporarily, it undoubtedly hastens the fatal termination.

DISEASES OF THE MAXILLARY ANTRUM OR SINUS.

Diseases of the maxillary antrum are by no means so rare as has hitherto been supposed, and this is probably due to the fact that the antrum has not always been carefully examined. The diseases most frequently met with here are mucocele and empyema, the latter being the more common of the two. The subjective symptoms are practically the same in both, and are often modified by coexistent nasal disease, such as polypi and hypertrophy.

Empyema of the Antrum.—Pus may collect in the antrum as a consequence of catarrhal inflammation extending from the nose, or it may originate in the antrum from decomposing mucus. The patients usually complain of pain in the region of the upper jaw, and if the collection of pus be considerable it may flow into the nasal meatus through the ostium maxillare, and out through the nostril or back into the pharynx. The most common source of purulent inflammation here is disease of the teeth, especially of the posterior molars, forming either subperiosteal abscess or

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abscess of the antrum itself. Another form of disease met with in the antrum is polypoid cysts, which may originate here, but are much more likely to arise in the naso-pharynx and involve the antrum secondarily.

Symptoms.—Pain is the most constant symptom, though if drainage be free it may be slight or entirely absent. It is located in the cheek, is frequently periodic in character, and is sometimes accompanied by an unpleasant odor from the decomposition of retained secretions. The escape of pus from the antrum is positive evidence of disease. Owing to the comparative proximity of the openings of the frontal, ethmoidal, and maxillary sinuses in the nose, it is necessary to exclude disease of the first two sinuses before we can make a positive diagnosis of disease of the antrum. Owing to the ostium maxillare being the lowest of these three openings, the antrum may frequently become the receptacle for pus coming from the other sinuses, which is guided into the antrum by a semilunar fold of mucous membrane in the nose. In addition to washing out the antrum through the ostium maxillare, we are sometimes aided in our diagnosis by employing the method of transillumination recommended by Ziem, Davidsohn, Burger, Caldwell, and others. The exploratory operations advised by Ziem and Lichtwitz are not necessary, as a diagnosis can generally be arrived at by careful examination, irrigation, and transillumination. For the purposes of examination by illumination of the antrum, the patient must be in a dark room. A four- or five-candle-power lamp, connected with a battery and a rheostat by means of ordinary insulated wires covered with rubber tubing, is placed in the mouth, the lips are closed over the connecting rubber tubes, and connection is made. If the antrum is normal, the face takes on a ruddy glow, the brightness differing in spots according to the thickness of the solid parts of the face. The brightest spot will be at the lower margin of the orbit, where the wall is thinnest. If the light encounters any abnormal substance, like mucus or pus, or polypi, or solid tumors, the illumination will be affected, even to more or less darkening of the corresponding side of the face. It is always advantageous to compare the two sides of the face in every case, as the contrast between a normal and a diseased sinus is generally very marked. As the floor of the nose, unless diseased, transmits light clearly, it will be well to continue this examination in the nose, for if the outer wall is in shadow, disease of the antrum is certainly present.

In cases of unilateral rhinitis, Burger advises to examine for suppuration of the antrum on the opposite side. He attaches much more importance to the illumination of the eye by transillumination, the signs of which are a red glow from the pupil and a subjective luminous sensation. He gives the palm to Heryng and Vohsen for exploiting this method of examination. He states that in many instances the cheek has been illuminated in spite of the presence of pus in the antrum, as subsequently discovered. When the mouth is illuminated, and a red glow is at the same time seen in the pupil, while the patient experiences a luminous sensation, the antrum must be normal, for all light passing from the mouth to the eye must pass

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through the bottom of the antrum. On the contrary, the cheek may be illuminated from the lamp in the mouth, even when there is pus at the bottom of the antrum, by light passing from the mouth to the nose and across the floor and through the outer wall of the nose, while the layer of pus at the bottom of the antrum prevents all illumination of the eye. In all these cases the failure to illuminate the eye may be due not only to empyema, but to any other form of disease of the antrum. Fuller reports and more extensive experience with this method of examination will doubtless enable us to judge more accurately of its value as an aid to diagnosis of disease of the antrum.

Treatment.—The only method which promises a cure is to open the antrum, carefully remove its contents, employ frequent irrigation with an antiseptic solution until the pus ceases to be secreted and the antrum has regained its normal condition, and then endeavor to close up the artificial opening. Several methods of operating have been employed, but the one which on the whole gives the best results is that recommended by Mr. Christopher Heath. This consists chiefly in making a large opening into the antrum through the canine fossa, through which the pus may be freely evacuated, cheesy masses and polypi and necrosed bone-fragments removed, and the whole interior of the antrum thoroughly curetted. Free irrigation is then to be employed as long as any pus comes away. At the same time the ostium maxillare may be enlarged or a new opening made through the outer wall of the nose into the antrum; and this opening is to be maintained, while that in the mouth may subsequently be allowed to close.

These cases of empyema are apt to be of long standing, and, unless the nasal meatus is kept healthy, they are obstinate in healing, as any opening into the antrum from the mouth is liable to close.

Tumors of the Superior Maxilla and Maxillary Antrum.—Pathological new growths of the superior maxilla differ in their clinical appearances according as they start from the alveolar arch or from the body of the bone. The former are of course visible in the mouth. Tumors of the antrum are difficult to recognize if they have not already caused distention of the sinus. The symptoms are pain in the teeth of the upper jaw, a dull pain in the region of the antrum, discharge of pus and blood from the nose in lying down, and more or less epiphora. The pain, which may be in the region of distribution of the infra-orbital nerve, is not apt to appear until the tumor has attained a considerable size and has more or less completely filled the antrum, the distention of the walls of the cavity causing the pain by pressure on the nerve-twigs. As the tumor grows, the walls of the antrum are gradually absorbed, and a new thin scale of bone is developed from the periosteum. This may occur in the anterior wall, or in the orbital wall, or in the alveolar wall, and the tumor soon extends towards the nose, and causes great enlargement of the opening communicating with the nasal meatus. These nasal growths extending from the antrum are often mistaken for nasal polypi.

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Finally, the growth frees itself from its bony envelope and comes to lie immediately under the soft parts of the cheek, and the diagnosis is rendered easier, either by a projection forward through the anterior wall of the antrum, or by displacement of the eyeball upward and outward, or upward and inward, by the protrusion of the floor of the orbit. If a swelling appears simultaneously in all the above places, a diagnosis may be made of tumor of the antrum. Protrusion of the anterior wall of the antrum alone might mean a cyst of one of the tooth cavities or a periosteal tumor, as well as a tumor of the antrum. In such cases puncture with a trocar would probably differentiate between an external and an internal tumor. A large tumor of the antrum would probably increase the breadth of the cheek, and would push the nose towards the opposite side. If the tumor grows from the bone itself, the inferior orbital margin is decidedly broadened.

Tumors from the antrum itself rather tend to break through into the nose, mouth, or orbit. Tumors of the superior maxilla, whether they start from the bone or from the antrum, gradually extend into all the neighboring cavities. They early involve the nasal meatus, thence extend into the spheno-maxillary and palatine fossæ and pharynx, and finally perforate the base of the skull. They usually involve the orbit later, and sometimes extend into it from the ethmoid cells, even before the floor of the orbit is perforated. In no case is it possible to diagnosticate a tumor of the maxillary antrum early in its development.

The *treatment* of these tumors consists either in extirpation of the growth or in complete resection of the superior maxillary bone.

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DISEASES OF THE EYELIDS.

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So many varieties of tissue are represented in the eyelids, several of which are more or less involved in most of the diseases that occur in them, that it is difficult to classify these diseases accurately. They may be conveniently studied, however, as they affect chiefly the skin, the subcutaneous tissue, the cartilage, the lid-margin, or the muscles.

Erythema occurs as the result of local irritation or as a symptom of disturbance of the circulation due to such causes as indigestion, dentition, uterine affections, etc. Idiopathic palpebral erythema is rare. The symptoms are a slight superficial redness, a kind of blush which disappears under pressure by the finger, and sometimes just perceptible swelling. There is usually no discomfort, but sometimes there is a slight burning sensation. The affection is generally transient, disappearing of itself in a few days, but occasionally it is more persistent and recurrent. The treatment must be directed chiefly to removal of the cause. Sedative applications may be of use in acute cases; in chronic cases it has been recommended to modify the local circulation by the use of compresses with acetate of lead, the application of solutions of nitrate of silver, etc.

Eczema of the delicate and sensitive skin of the lid is a distressing affection. It occurs in the same forms and from the same causes as upon the skin elsewhere. The swelling is greater than in other localities, and may be sufficient to close the eye. In the treatment, weaker applications should be used than are readily borne in other parts. In the acute stage soothing or slightly astringent remedies are required, such as glycerite of starch, carefully prepared diachylon ointment, oxide of zinc and acetate of lead cerates, etc., or dusting with starch powder, alone or mixed with powdered oxide of zinc in the proportion of one part of the latter to three or four parts of the starch. Aristol has been highly recommended. When crusts form, it is useless to employ any remedy before their removal, which is accomplished by means of soaps or alkaline washes or ointments. In the chronic form more stimulating remedies are required. One of the most efficient is carbolic acid, which relieves the itching. Five grains may be added to an ounce of zinc ointment. Mercurials are sometimes useful. The condition of the general health will often call for laxatives, antacids,

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or chalybeates. Some authors recommend arsenical preparations in the chronic forms of the disease.

Herpes Zoster Ophthalmicus is a neuropathic affection due to disease of branches of the fifth nerve or of the Gasserian ganglion. It may involve one or more or all of the branches of the ophthalmic, and the vesicles occur along the course of the affected nerves. The neighboring skin becomes red and swollen, presenting a close resemblance to erysipelas, for which this disease is very often mistaken. The symptoms are limited to the distribution of the filaments of the nerve affected, and never extend beyond the median line unless both sides are simultaneously involved, which rarely happens. There are generally severe pain and considerable febrile reaction. Neuralgia may precede the eruption for some hours, days, or even weeks, and is in most cases intense, but in a few is almost wanting. Patients sometimes continue to suffer pain for months or years after the eruptive disease has subsided. There is partial or complete anæsthesia of the region of skin affected, which may continue after the subsidence of the pain and inflammation. In milder cases the vesicles disappear without leaving any mark, but in the more severe they become pustular and may result in sufficient destruction of tissue to leave permanent scars. The conjunctiva, cornea, and iris are frequently involved in the disease, and a number of recorded cases were complicated with paralysis of one or more of the orbital muscles. Of two cases occurring recently in my own practice, one had complete ptosis and the other had paralysis of all the external muscles supplied by the third pair, without mydriasis. A case reported by Haltenhoff[1] was complicated with extensive retinal hemorrhage.

The etiology of this disease is obscure and its treatment is unsatisfactory. Local applications have little or no curative effect. Internally, anodynes will nearly always be required. Large doses of quinine have seemed useful in some cases, and treatment by galvanism is recommended for the relief of persistent neuralgia and paralysis.

Erysipelas of the eyelids is of frequent occurrence, usually in connection with facial erysipelas. The delicacy, distensibility, and fulness of the skin and the looseness of its attachment predispose to great swelling from serous exudation, which makes it impossible for the patient to open the eye and difficult for the surgeon to inspect the ball. The superficial form is attended with little danger, but phlegmonous erysipelas may extend to the capsule of Tenon and the orbital tissue and result in blindness from optic neuritis or atrophy from pressure. A fatal result may occur from extension of the inflammation along the optic-nerve sheath to the meninges, though this is rare. Sometimes contraction of an abscess-cavity causes serious retraction of the lid, or sloughing of the skin produces an ectropion.

The treatment must be conducted on the same principles as in erysipelas elsewhere. The painful burning sensation is relieved by protecting the sur-

[1] Annales d'Oculistique, tome cix. p. 261.

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face of the skin from contact with the air by dusting it with rice flour or oxide of zinc powder. In a high grade of superficial inflammation some authors advise the application of iced cloths. A great number of local applications have been recommended; recently ichthyol has been highly praised. If suppuration or sloughing threatens, warm fomentations should be used and free incisions may be necessary.

Rhus Poisoning is a frequent cause of a violent inflammation of the skin of the lids in connection with that of the rest of the face (*dermatitis venenata*). It is the result of contact with the "poison ivy" (*Rhus venenata*) or with the "poison oak" (*Rhus toxicodendron*). The skin is of a deep-red color, and the subcutaneous connective tissue is œdematous. The lids are swollen and puffy, and in severe cases the eyes are completely closed. There are patches of vesicles, sometimes confluent, exuding a yellowish fluid which on drying forms a thin soft crust. The first symptoms are burning and itching, which frequently become intense.

A number of other plants produce a similar disease in a few persons exceptionally susceptible. I met recently at the Wills' Hospital with an almost typical case of it which resulted from the application to the eyes of a solution of sulphate of duboisine for purposes of refraction. The symptoms commenced a few hours after the first application. There was some congestion of the conjunctiva, but the affection of the skin was entirely different from the erythematous inflammation occasionally met with in connection with conjunctivitis produced by the prolonged use of atropine. A great number of remedies have been lauded as specifics in this affection, but they owe their reputation to the fact that the disease is self-limited and its acute symptoms subside in a few days. Dilute lead-water is the most soothing application. Acetate of morphine may be added.

Chromidrosis (colored sweat) may occur on any part of the skin, but the place of election is the lower eyelid. There is a dark-blue or blue-black coloration, which is easily removed by friction with any oily lotion but soon returns. It has been known to last for several years. The skin appears normal. Nearly all the patients have been women, most of whom have been the subjects of menstrual irregularities, and the condition is so often simulated that its existence as a genuine disease has sometimes been doubted; but it has been definitely established. Indigo, plumbago, and soot have been detected in feigned cases. The pathology of the disease has not been determined. It is usually considered an affection of the sweat-glands, but some authorities think it has its origin in the sebaceous glands. Various astringent and stimulant applications have been recommended.

Xanthelasma, or **Xanthoma**, is an affection occurring much more frequently on the eyelids than elsewhere, though it has been met with on the skin of the face, flexures of the joints, hands, feet, etc., and in mucous membranes; and Virchow has described a case in which it was found upon the cornea. It appears in the skin of the eyelids as small, smooth, yellowish patches, slightly raised, and usually of an irregular oval form. It may be

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single or multiple, is found most frequently near the nasal angle of the upper lid but may occur in any part of either or both lids, and is sometimes symmetrical on the two sides. It occurs in elderly persons, and is said to be more common in women than in men and on the left side than on the right. It is very slow of growth and innocent in character. The disease consists in fatty degeneration of hyperplastic connective-tissue corpuscles. (Waldeyer.) If cosmetic considerations demand the removal of the patches, they are easily excised. Noyes has found the disease to return after removal, in one case.

Milium is a superficial, small, round, pearly elevation about the size of a millet-seed, from which it derives its name. It consists of an accumulation of the contents of a sebaceous gland whose duct has been obstructed. It should be punctured with the point of a cataract-knife and evacuated.

Molluscum Epitheliale (*molluscum contagiosum*) is a small, rounded, dingy-white tumor, sometimes attaining the size of a pea, though usually smaller. The top is flattened and presents a dark spot in the centre representing the aperture of a follicle. It contains a cheesy mass, which under the microscope is found to consist of degenerated epithelial cells and albuminous corpuscles resembling swollen grains of starch and called molluscum corpuscles. It has its origin in the transformation of a sebaceous gland. It was formerly considered to be contagious, but this view is now generally abandoned.

The treatment is simple. It is sometimes sufficient merely to press out the contents with the finger-nails or an entropion forceps; or it may be necessary to cut open the tumor and excise the walls of the cavity, or to cauterize it with nitrate of silver.

Malignant Pustule may occur upon the lids. It is rare in this country. It is usually considered a specific affection resulting from inoculation with a virus germinated by cattle suffering with the murrain. This poison is said to be retained for a long time in the skin and hair of animals that have died of this disease. According to some authors, malignant pustule may be produced by contact with decomposing animal matter. It commences in the skin as a small vesicle surrounded by an areola, and subsequently the subcutaneous tissue is involved, forming a hard circumscribed base. The vesicle soon bursts, and is followed by a sloughing ulcer which spreads rapidly in all directions. There are severe constitutional symptoms, such as nausea, fever, chill, prostration, and delirium, and death often results from blood-poisoning.

The treatment is the same as for malignant pustule occurring elsewhere, except that the caustic potassa usually recommended cannot well be applied to the lid, and the thermo-cautery should be substituted.

Gangrene.—Noyes[1] refers to several cases of spontaneous gangrene of the skin of the lid; and Wecker[2] describes, under the name of "malignant

[1] Diseases of the Eye, p. 233.

[2] Wecker et Landolt, i. p. 46.

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œdema," a diffuse form of phlegmonous inflammation with a tendency to gangrene in large patches, without the formation of a pustule.

Phlebitis.—The lids may be involved with neighboring parts in facial phlebitis. This affection is liable to result fatally from extension to the cavernous sinus.[1]

Lupus.—The ulcer of lupus vulgaris is occasionally found upon the eyelid, generally extending there from the nose or the cheek, particularly from the former. It is said to be extremely rare in this country. Van Harlingen has never seen it in a native American. It is liable to be confounded with syphilitic ulcer or epithelioma. (See Epithelioma.) Scraping with a sharp curette and dusting with iodoform, and destruction by the actual cautery, are among the most efficient methods of treatment.

Chancre.—The primary syphilitic sore has been occasionally met with upon the eyelid, and the possibility of its occurrence should be borne in mind. The preauricular glands are indurated. Its usual seat is near the lid-margin, but it is more likely to extend in the skin than to involve the conjunctiva. The cartilage generally escapes.

Secondary Syphilitic Ulcer may occur from the breaking down of a tubercle of the skin, or of a gumma originating in the skin or more frequently in the subcutaneous tissue and cartilage. Its most frequent seat is in the skin near the lid-margin or below the inner canthus. In rare cases it has occurred on the conjunctival surface of the lid. It is a late manifestation, and, though usually classified as secondary, might perhaps be more correctly placed among the tertiary lesions. The fact that this ulcer is sometimes met with long after other syphilitic symptoms have subsided adds to the difficulty of diagnosis. It may be mistaken for lupus or epithelioma, particularly for the latter, from which it is sometimes difficult to distinguish it. (See Epithelioma.) When situated over the lacrymal sac it has been taken for dacryocystitis. (Mackenzie.) A prompt diagnosis is important, as, while the syphilitic ulcer usually yields promptly to proper constitutional treatment, caustics are useless and the knife is dangerous.

Epithelioma occurs more frequently on the eyelids than elsewhere. Its usual seat is near the margin of the lower lid, particularly at the inner canthus, where it extends to the angle of the upper lid also. The superficial form commences in the upper layers of the skin as a flat, wart-like elevation, or plaque, whose surface becomes excoriated and secretes a watery, viscid, or sometimes sanious fluid which forms a brownish crust. Beneath this crust appears a superficial grayish excavated ulcer with a slightly raised base surrounded by induration. The progress is extremely slow, sometimes extending over a long series of years without serious inconvenience to the patient. The lymphatic glands are not usually indurated.

The **Rodent Ulcer**, which is classified with this form of epithelioma,

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[1] Blachez, Gazet. Hebdom., October 30, 1863, and Dubreuil, ibid., November 30, 1863.

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spreads slowly but extensively, and attacks any tissue that is met with in its course.

The deep-seated form of epithelioma commences as a tubercle the size of a split pea, involving the skin and connective tissue. The little tumor breaks down, forming a deeply excavated ulcer about which the skin is infiltrated and congested. It is a much more serious affection than the superficial form, progresses more rapidly, returns more certainly after removal, and extends into the orbit, attacking the periosteum and conjunctiva. The lymphatic glands may become indurated in the later stages.

Epithelioma is liable to be mistaken for lupus or syphilis. It is a disease of advanced age, and is not often met with in persons under forty. It is much slower in its progress than syphilis, but not so slow as lupus. The ulcer is slightly raised, its edges are hard and everted, it is surrounded by a circumscribed induration, and often bleeds easily. Lupus is a disease of early life, usually commencing before puberty and progressing very slowly. The ulceration is superficial, and the surrounding induration is moderate and diffuse. The border of the ulcer is soft and not sharply defined, and is often undermined. There are generally a number of points of ulceration which become confluent. Chancre is more likely to be met with in youth or middle life. Its course is acute, and there is early induration of the preauricular glands. Other syphilitic symptoms or a history of infection will often make the case clear. The late syphilitic ulcer is more rapid in its course than the epitheliomatous, and usually occurs in younger subjects, most frequently in middle life; its edges are not so hard, and it is not surrounded with induration. In doubtful cases the therapeutic test of mercury and the iodides should always be resorted to.

The treatment of epithelioma consists in destruction by caustics or removal by the knife. The former method is adapted to superficial ulcers of small extent, or to cases of more extensive disease in which excision is for any reason impracticable. Caustic potash and the various destructive pastes or ointments used elsewhere are not applicable to the eyelids. Nitric or chromic acid is efficient. I have found chloracetic acid the most satisfactory. It is applied in saturated solution, by means of a little pledget of absorbent cotton on the end of a probe or wooden toothpick, to the whole surface of a small ulceration or about the edges of a larger one. It is followed by but slight reaction and causes very little pain, and may be frequently repeated if necessary. The galvano-cautery is a favorite with some surgeons. Others scrape away the diseased tissue with a sharp spoon or a scalpel. Strong claims have been made for a saturated solution of chlorate of potash. Cases of considerable extent are best treated by free excision. (See Blepharoplasty.)

Variola.—The pustules of small-pox sometimes result in serious injury to the lids. When confluent and deep, they may cause considerable loss of substance of the skin, followed, after cicatrization, by ectropion. If they form upon the lid-margin, they involve the Meibomian ducts and the bulbs

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of the cilia, and may cause obstinate marginal blepharitis, loss of the lashes, or trichiasis.

Treatment.—Among simply soothing applications, which perhaps act chiefly by protecting from contact with the air, are cosmoline, albolene, glycerite of starch, etc., and dusting with rice flour or oxide of zinc powder. Aristol powder has been suggested. Mercurial ointment spread on lint is a favorite with some surgeons, and yellow oxide of mercury ointment (gr. i to 3i) may be applied frequently to the lid-margins. Boroglyceride (fifty per cent. in glycerin) is soothing and antiseptic and a good protectant. Ichthyol, in fifty-per-cent. ointment or wash, has been highly recommended for the prevention of pitting, and may be useful in these cases. It would probably need further dilution for use upon the delicate skin of the lid. In a high grade of acute inflammation with much swelling, iced cloths may give most relief. The conjunctivitis that usually complicates the affection of the lids aggravates it by the irritation that the discharge produces, and the conjunctival sac should be frequently cleansed by free douching with boracic acid wash.

Vaccinal Eruption also occasionally occurs upon the eyelids, but it is not likely to do serious mischief. There is considerable swelling of the lid and enlargement of the preauricular gland.

Warts, originating in hypertrophy of the papilla of the skin, may occur on any part of the lid, generally near the margin. They can be easily removed with scissors or fine ligature.

Horny Growths (*cornu cutaneum*) have been occasionally met with on the lids. In several cases they have acquired considerable size. The base should be cauterized after removal.

Angioma.—Several forms of angioma are met with in the eyelids. The *simple vascular nævus*, or "mother's mark," appears as a bright-red or a "port-wine-colored" patch of skin which may be the size of a pin-head or large enough to cover the whole lid. It is level or slightly raised, and pales temporarily under pressure. It is the result of excessive development of capillaries, and is always congenital, occasionally disappears spontaneously, but is usually permanent and rarely increases.

Telangiectasis (dilatation of distant vessels) consists of a collection of enlarged capillaries, arterioles, and venules in the skin and the subcutaneous tissue, sometimes chiefly in the latter, which may exist at birth but generally appears later, and in either case has a tendency to increase.

Cavernous Angioma forms a distinct tumor consisting of cells and sinuses and enlarged vessels with a framework of connective tissue. It also may be congenital, but more frequently originates after birth, and is sometimes developed from the preceding variety. Vascular tumors of the lid become turgid when the head is held down, or, in the case of children, when the patient cries. They can generally be emptied by pressure, but occasionally pulsate if chiefly arterial in character and offer greater resistance to pressure.

Treatment consists in obliteration of the vessels, destruction by cauteri-

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zation, or removal with the knife. Small superficial nævi may be successfully treated by caustics, such as ethylate of sodium and nitric, chloracetic, or chromic acid, applied by means of a glass rod or a little pledget of cotton twisted about the end of a fine probe, or by the actual cautery. In the case of infants, vaccination at several points, or by means of a thread saturated with vaccine matter and passed across beneath the skin, will often excite sufficient inflammation to obliterate the vessels. A larger growth may be attacked by penetrating a fine thermo-cautery needle obliquely under the skin at numerous points around its base. Coagulating injections are not without danger, and ligatures are likely to cause suppuration and leave cicatrices: when injections are used, the part should be enclosed in a Snellen clamp. Cavernous angiomata are frequently encapsulated, when they can be safely enucleated. In other cases removal should be accomplished by incisions carried well into the sound skin. A blepharoplastic operation may be necessary. For extensive growths, particularly those extending into the orbit, electrolysis is recommended.[1]

Lepra.—The eyelids may be involved in leprosy of the face.

Elephantiasis.—True elephantiasis of the eyelids is extremely rare. A few cases have been reported.[2] It consists of an hypertrophy of the skin and subcutaneous connective tissue, and may form tumors of considerable size.

Lymphangioma.—Rare instances of this disease in the eyelids have been recorded, usually in connection with a similar growth in the orbit. Dr. Dunn[3] has reported one case and collected several from literature. In Chauvel's case[4] the orbit was not involved. There was a large tumor of the upper lid completely closing the eye. It was hard and elastic and closely adherent, and was composed of tissue similar to that of lymphatic glands. There are always other symptoms of leukæmia. Michel[5] describes, under the name of *circumscribed lymphangioma*, a reddish, smooth, partly translucid tumor, the size of a pea, and containing a clear liquid, situated at the inner angle of the lid-margin. The microscope showed a cavernous tissue infiltrated with lymphoid cells.

Œdema.—The delicacy, distensibility, and fulness of the skin and the looseness of its attachment render the lids peculiarly liable to serous infiltration. Œdema is frequent as a result of local congestion in the lids themselves, the conjunctiva, or the orbit, or of disturbance in the general circulation. It is often an important symptom of disease in distant organs, particularly affections of the heart and kidneys, arsenical poisoning, and trichinosis. No other treatment than removal of the cause is usually required. If the swelling is sufficient to interfere with the opening of the

[1] Nieden, Arch. d'Ophtalmol., t. x., 1, p. 26.
[2] Wecker et Landolt, tome i. p. 88.
[3] Trans. College of Physicians of Philadelphia, vol. xv. p. 103, 1893.
[4] Gaz. Hebd., No. 23.
[5] Graefe u. Saemisch, Bd. iv. S. 422.

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eye, evacuation of the serum by puncture and the application of pressure will give relief.

Ecchymosis.—The most common cause of extravasation of blood in the subcutaneous tissue is a direct contusion, as in the case of the "black eye" resulting from a blow of the fist. It appears immediately after the injury. When the result of a contusion of the forehead or temple, ecchymosis invades the lid by the external canthus. An effusion of blood appearing more gradually, after an injury of the head, and commencing in the orbit and beneath the ocular conjunctiva, is considered by some surgeons as one of the most positive signs of fracture involving the orbital walls or the base of the cranium. Spontaneous ecchymosis is rare. It may occur in scurvy, or as a result of weakening of the vessels from old age or other cause, or may be induced by violent straining in whooping-cough. True *hæmatoma*, forming a circumscribed tumor, is not often met with in the lids, on account of the looseness of the connective tissue. Immediately after the injury the application of ice, or of cloths wet with iced water, is the best means of checking the effusion. If extensive swelling has occurred, compression with a rather firm bandage promotes rapid absorption. Slightly stimulating embrocations, among which tincture of arnica has enjoyed a special reputation, are recommended.

Emphysema of the lids is produced by a communication between the subcutaneous tissue and neighboring air-cavities, usually the lacrymal, nasal, frontal, or maxillary. Fracture of the base of the skull involving the sphenoidal or the ethmoidal sinus is a possible cause. It is nearly always caused by external violence, but may occur without traumatism, as the result of an opening formed in the bone by disease. In either case the air is forced into the connective tissue by the expiratory effort in blowing the nose, the avoidance of which is generally the only treatment needed. Compression may sometimes be of use.

Abscess of the Lid is generally traumatic and most frequently the result of contusions. It may have its origin in caries of the orbital margin. According to Berger,[2] deep abscesses of the upper lid which have been observed as a result of influenza may have been due to inflammation of the frontal sinus. Spontaneous abscess is rare in adults, but more frequent in badly nourished children. Large abscesses, particularly if not promptly opened, sometimes cause sufficient sloughing to produce considerable deformity of the lid with lagophthalmos or ectropion.

There is at first diffuse redness with swelling, much like that seen in erysipelas, but soon a hard spot can be detected which increases in size and finally softens in the centre. The whole upper lid may be involved and present an extensive swelling. An abscess situated in front of the lacrymal sac closely simulates dacryocystitis.

Treatment.—Phlegmonous inflammation may sometimes be relieved in

[1] Maladies des Yeux dans leurs Rapports avec la Path. gén., p. 194.

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its early stages by the application of iced cloths, which, at any rate, will be agreeable to the patient; but so soon as decided induration is detected, warm fomentations or hot stupes should be applied. When evidences of pus appear, a free horizontal incision should be made at once, and if a considerable cavity remains it should be syringed with an antiseptic solution.

Furuncle is a localized inflammation of the skin accompanied by a small gangrenous slough of the subcutaneous tissue,—practically anthrax on a small scale. Some authors make the distinction that in the latter the skin is more involved in the gangrene. In old and debilitated subjects the disease may assume a form that would be generally denominated anthrax. The French give the name of "bouton d'Alep" to a form of furuncle occurring epidemically in hot countries and supposed to be of microbic origin.

Hordeolum, or "stye," is sometimes described as a small furuncle occurring about a hair-follicle on the lid-margin. (Wecker.) It appears as a hard, sensitive nodule, not usually larger than a grain of barley (*hordeum*), on the anterior lip of the edge of the lid. The redness and swelling of the skin about it may extend over the entire lid, and the conjunctiva is congested. The Meibomian glands are not involved. Owing to the close structure of the cartilage and the great sensitiveness of the lid-margin, the pain is comparatively severe. Usually a hordeolum runs an acute course and ruptures in a few days, giving exit to a small slough consisting of degenerating pus-corpuscles, altered fat-globules, epithelial detritus, and connective tissue. Occasionally the inflammation is aborted and the contents of a small stye are absorbed, or it assumes a chronic form and the little lump remains for a long time ("blind stye"). The inconvenience of a single hordeolum would be a small matter, but styes are apt to be multiple, are nearly always recurrent, and are an indication of some disturbance of the general health. An almost continuous series is not uncommonly kept up for weeks or months. Exposure to heat or dust, or to any form of local irritation incident to the occupation of the patient, and the accommodative strain of uncorrected optical defects, are exciting causes.

Treatment.—In the acute form warm fomentations or hot stupes give most relief and hasten the process of suppuration and rupture. When the swelling points, it should be punctured with a Beer or a Graefe knife. Occasionally an indolent ulcer or a sinus remains after evacuation, and requires the application of the pure or the mitigated nitrate of silver pencil. When suppuration does not occur, resolution is promoted by the application of nitrate of silver solution, gr. x to ℥i, and the use of ointment of yellow oxide of mercury. The ointment is also sometimes useful in preventing the tendency to recurrence. All sources of local irritation should, of course, be removed so far as possible, and careful attention should be given to the general health.

Lipoma is very rare in the eyelids, whose connective tissue is normally devoid of fat. According to Panas, it is usually found between the muscle

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and the tarsus. In four cases reported by Schell[1] it was found between the skin and the muscle. It may involve the whole lid, or may form a circumscribed tumor. It is soft and elastic to the touch and lobulated. The skin over it is mobile, and it is easily excised.

Hernia of the Fatty Tissue of the Orbit is described by Wecker.[2] This tissue is normally retained in place by the skin, the orbicular muscle, and the tarso-orbital fascia. When this support is weakened by the atrophy of age or partially ruptured by traumatic injury, the orbital fat, particularly if developed in excess, may appear in pouches bulging the distensible skin. These hernias occur only on the lower lid, as the upper tarso-orbital fascia is reinforced by the insertions of the levator tendon and is much more resisting than the lower. There are two forms of this hernia, the senile and the traumatic. In the former, relaxation of the tarso-orbital fascia permits a hernia of the fat, which separates the atrophied muscular fibres and appears beneath the flabby skin. In the latter, which may occur in persons of middle age, the hernia is due to a rupture of the tarso-orbital fascia by some violent strain, as in coughing, or by a blow.

These tumors are not compressible, but are freely movable and easily pushed back into the orbit, which distinguishes them from simple relaxation of the skin with œdematous infiltration.

If the deformity is sufficient to demand surgical interference, it can be removed by a simple operation. The lid is made tense by means of a horn spatula in the conjunctival sac while an incision passing through the skin, some muscular fibres, and a thin envelope of connective tissue, frees the nodule of orbital fat. This nodule is seized with forceps and cut off close to the margin of the orbit.

Fibroma.—Several forms of fibroma are mentioned by authors as occurring in the eyelids. They are developed in the fibrous tissue of the deep layers of the skin or of the subcutaneous or submucous connective tissue. The "painful subcutaneous tubercle" is a small firm circumscribed nodule, situated in the connective tissue immediately under the skin, which is movable over it and is normal in appearance. These little tumors are painful and sensitive to the touch. They were first described by Wood in 1812,[3] and have been frequently studied since, but the presence of nerve-fibrils has never been demonstrated. (Sutton.) Fibromata of the lid sometimes assume the form and consistence of plates of cartilage. In a case observed by Wecker,[4] when the lid was everted, a tumor two centimetres long and one centimetre wide projected from the cul-de-sac and presented the appearance of an additional cartilage. Von Graefe[5] has described a tumor, also situated in the cul-de-sac, which contained true bone.

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[1] Transactions American Ophth. Soc., vol. iv. p. 49.
[2] Le Progrès Médical, 19 Mars, 1892.
[3] Edin. Med. and Surg. Journ., p. 283.
[4] Wecker and Landolt, vol. i. p. 93.
[5] Klin. Monatsbl. f. Augenheilk., January, 1863.

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Fibroma molluscum involving the skin and the subcutaneous connective tissue may form an extensive pendulous tumor in the loose and distensible integument of the lid. One reported by Horner[1] was nearly as large as an egg.

Fibromata are usually present, in small size, at birth, and sometimes assume a rapid growth after remaining nearly stationary for years. When the skin is involved, removal of a tumor may be attended with considerable difficulty and necessitate extensive plastic procedures.

Neuroma has been met with in the eyelids, usually in connection with similar tumors in other parts. It consists of degenerated nerves and dense hyperplastic connective tissue, and forms tortuous nodosities which give the impression of cords rolling under the fingers and have been compared to a mass of earth-worms. It is congenital, and increases with the growth of the subject.[2]

Cysticercus.—A number of cases have been recorded in which a cysticercus arising in the conjunctiva or the orbit formed a tumor of the lid by extending forward. Sichel[3] has reported one example in which it was found between the skin and the tarsus.

Tarsitis as a distinct affection is very rare. The tarsus, on account of its close connection with the conjunctiva, participates in the chronic inflammations of the latter. The atrophy and distortion of the cartilage in connection with cicatricial contraction of the conjunctiva after trachoma is an important factor in the production of entropion. The histological changes found in the tarsus in these cases have been described by Michel.[4] Deep inflammation of the skin, particularly of the erysipelatous form, may also extend to the cartilage.

Inflammation originating in the tarsus is nearly always syphilitic. Its progress is very slow. The swelling is sometimes considerable and is hard to the touch, and the skin moves freely over it. Amyloid degeneration of the hypertrophied cartilage has been observed.[5] The syphilitic form of tarsitis usually yields to appropriate specific treatment. In addition to the internal administration of mercurials and iodides, mercurial ointment may be applied locally. Hot stupes are also useful. Changes in the form of the cartilage produced by inflammation frequently require operative treatment.

Hypersecretion of the Meibomian Glands forms crusts on the lid-margins. The secretion may accumulate in the glands or ducts and give rise to small abscesses, or to calcareous concretions (palpebral lithiasis) which appear as little yellowish nodules on the conjunctival surface easily picked out with a needle or the point of a Graefe knife.

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[1] Klin. Monatsbl. f. Augenheilk., Bd. ix. S. 1.
[2] Panas, Traité des Maladies des Yeux, tome ii. p. 117.
[3] Rev. Méd.-Chir. de Paris, tome i. p. 224.
[4] Graefe und Saemisch, Bd. iv. S. 445.
[5] Wecker, vol. i. p. 133.

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Chalazion is a small tumor situated in the tarsus. It is tense and rounded and is firmly adherent to the cartilage, but the skin moves freely over it. Its contents are gelatinous or, in the later stages, may be purulent. It originates in inflammation of a Meibomian gland, but is not, as was formerly thought, a retention cyst. The process seems to consist of hyperplasia of epithelium, proliferation of connective tissue, and retention of secretion. A mass of granulation tissue is formed, for which Virchow proposed the name of granuloma. This tissue undergoes degeneration and breaks down. An imperfect cyst-like wall is formed by condensation of the surrounding tissue. The microscope shows a number of small round cells, fewer giant cells, and some fusiform cells enclosed in a gelatinous substance.

The progress of a chalazion is very slow. If not removed, it usually forms close inflammatory adhesion to the conjunctiva which finally gives way and allows the softened portion of the mass to escape, while granulations project through the fistulous opening. The skin may also become adherent. The position of a chalazion is shown on the under surface of the lid by a bluish or yellowish patch of inflamed and thinned conjunctiva. Small tumors occasionally disappear by resolution or, more rarely, are transformed into fibrous tissue. Chalazia are frequently multiple. Their etiology is obscure. Sometimes they appear to be caused by a chronic blepharitis, but not infrequently the lid is healthy, except at the seat of the affected gland. The treatment is essentially operative. Friction with mercurial or iodide ointments has been recommended to promote the resolution of small and recent tumors. The iodide of cadmium has the advantage of not staining the skin. These applications are harmless and may be useful, though they probably owe their reputation to the fact that spontaneous disappearance is not very rare. If there is blepharitis it should, of course, be carefully treated, and all sources of irritation, such as refractive errors and over-use of the eyes, should be avoided. The general health may require attention.

Sarcoma.—Primary sarcoma of the eyelid is rare. Zimmerman[1] has recently reported a case and referred to five others. In two of these six cases the origin of the disease was probably in a Meibomian gland, and in one in the tarsal conjunctiva. In one there was sarcomatous degeneration of a pigmented nævus, and all were more or less pigmented. A rare case of sarcoma of the tarsus and conjunctiva of the lid, with amyloid infiltration, has been reported by Prout and Bull.[2] In a case recorded by Van Duyse,[3] occurring in a child seven years of age, a myxo-sarcoma of the upper lid grew with great rapidity and reached the size of the patient's fist in a few months. The growth commenced in the connective tissue between the orbicular muscle and the tarsus. Van Duyse, in an interesting discussion

[1] Ophthalmic Review, vol. xiii., No. 152.
[2] Archives of Ophthalmology, 1879, p. 73.
[3] Annales d'Oculistique, 1887, tome xcviii. p. 112.

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of the subject, reviews a number of cases found in literature, and states that in most of them there was a history of rheumatism; in a small number the disease originated in the palpebral conjunctiva, and in the majority of these the tumor was pedunculated. Wilmer[1] reports a case and furnishes a bibliography of thirty-five. Altogether, forty-five cases have been recorded.

Sarcomatous tumors of the lid are more or less elastic to the touch and generally rounded, and the skin is movable over them. When situated in the cartilage they may, while still small, bear a very close resemblance to chalazia, which, however, they soon outgrow. The diagnosis is easier if they are pigmented. Sarcoma of the lid is generally characterized by rapid growth and a marked tendency to early recurrence; hence operative treatment should be prompt and radical.

Adenoma.—Cases of adenoma of the Meibomian glands and of the glands of Krause have been reported.[2]

Gumma.—Small gummatous tumors are occasionally met with in the lid, but they usually break down and appear as ulcers before they are brought to the attention of the surgeon. They are frequently multiple, and sometimes several little nodules coalesce. When seen before necrosis takes place, they bear a very strong resemblance to chalazia, but nearly always progress towards the skin and leave the conjunctival surface normal. They should not, of course, be attacked by the knife, but should be treated by the administration of mercurials and iodides.

Blepharitis Marginalis (Blepharo-Adenitis; Ophthalmia Tarsi).—Inflammation of the lid-margin may involve the skin, conjunctiva, cartilage, sebaceous glands, and, when it extends more deeply, the bulbs of the cilia and the Meibomian glands. It varies from simple hyperæmia to serious disease of all the tissues and ultimately complete destruction of the cilia, eversion of the lid-margin, and obliteration of the lacrymal punctum.

Hyperæmia of the lid-margin is often chronic and obstinate. The edge of the lid is congested and red and slightly swollen, and there is some increase in the secretion of the sebaceous glands. The eye is sensitive to bright light, cold, wind, or dust, and is irritated by close work. This irritability and the disfigurement that accompanies it occasion much annoyance, though the vision may remain perfect. This affection is more frequent in blondes and in persons of strumous diathesis. Over-indulgence in eating or drinking, or other excesses, and uterine derangements may be mentioned among its causes. In many cases it is the result of a reflex vascular disturbance induced by the strain that defects in refraction or in muscular balance cause, but in some it is a more or less constant and permanent condition without known cause and with little probability of lasting cure. In the milder form of true blepharitis the margin is more congested and swollen than in simple hyperæmia, and exfoliation of epi-

[1] Trans. Amer. Ophth. Soc., 1894.

[2] Rumschewitsch, Klin. Monatsbl., 1890, p. 396.

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thelial cells and excessive sebaceous secretion form scales and crusts which collect at the roots of the cilia. When there is more extensive inflammation, the profuse secretion of the sebaceous and Meibomian glands forms matted masses of dried discharge in the cilia, beneath which the edge of the lid is found to be excoriated, or ulcers appear at the roots of the lashes. Some of the cilia adhere to the crusts when they are removed, or are easily pulled out by the fingers, and may have a drop of pus on the diseased bulb. They are succeeded by imperfect cilia, which sometimes take a wrong direction and give rise to trichiasis. If the inflammation is intense and prolonged enough to obliterate the follicles, there is complete absence of cilia (madarosis). The final condition in the worst cases is a thickened, rounded, and everted lid-margin, with a smooth cicatricial surface on which the orifices of the follicles, gland-ducts, and canaliculus are lost (lippitudo).

Blepharitis is nearly always binocular. Persons with a tendency to disease of the sebaceous glands, as shown by the presence of acne, are predisposed to it. According to some authors it is, in the largest proportion of cases, of eczematous origin. It occurs most frequently in badly nourished and neglected children of the poor, and in them is often associated with phlyctenular ophthalmia and intertrigo. Conjunctivitis, particularly that following measles, and lacrymal obstruction are local causes.

In treatment it is of the first importance to remove all sources of irritation. Optical errors should be corrected by the constant use of proper glasses, which not only relieve the strain but are of use as a protection against wind and dust. If there is photophobia, a slight smoke tinge may be given to the glass. Close work should be restricted, when possible, particularly at night, and early hours enjoined. A cure will be effected by these measures alone in many mild cases, but will be promoted by applying to the edges of the lids at night an ointment of the yellow oxide of mercury (half a grain or one grain to one drachm of simple cerate or albolene) and painting them two or three times a week with a two-grain solution of nitrate of silver. An accompanying conjunctivitis may require attention, and if there is lacrymal obstruction it should be promptly and radically treated. In more severe cases it is useless to make any application until the crusts have been removed, which can usually be best accomplished by means of hot water and absorbent cotton. If the crusts are very thick and dense, they may be softened by adding bicarbonate of soda to the water and retaining the cotton for a while upon the eye by a bandage. Though this process is sometimes tedious, it is better than intrusting forceps to the patient's friends. The strength of the yellow oxide ointment and of the nitrate of silver solution may be increased to two grains to one drachm of the former and ten grains to one fluidounce of the latter. In eczematous cases oxide of zinc is a useful addition to the ointment. Panas recommends the iodide of mercury in olive oil, in the proportion of 2 to 1000, and claims that this application has an advantage over ointments in penetrating the hair-follicles. Some authors advise touching the ulcerated points with

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solid nitrate of silver, but this is likely to prove irritating in many cases, as is also too much diligence in epilation. Fresh air and pure surroundings are always desirable, though too often unattainable, and cod-liver oil, arsenic, and iron may be needed.

Canities, or **Poliosis.**—In this affection there is an absence of pigment in the cilia, without other recognizable lesion. It may affect all the lashes of both lids, or may be confined to a part of those of one lid. The eyebrows also are sometimes involved. Canities is in most cases congenital, but is more rarely of neurotic origin.[1] It has been observed in cases of sympathetic ophthalmia, as a result of a violent blow upon the forehead and in connection with exophthalmic goitre.

Alopecia is occasionally congenital, and there is usually complete absence of the lashes in general alopecia. More frequently it is the result of local disease. In rare cases permanent loss of all the lashes occurs, with little or no inflammation of the lids and without recognizable cause. Palpebral alopecia is a quite constant symptom in leprosy.

Phthiriasis Ciliorum is an affection of the lid-margin due to the presence of pediculi pubis in the lashes. It occurs usually in the lower class of hospital patients, and the insect is generally supposed to be transferred to the lashes from the pubis by the fingers of the subject. It is a question whether the pediculi ever appear originally in the lashes. They are found there sometimes in children before the growth of pubic hair, but in these cases may be derived from the pubes of older members of the family. Occasionally the affection is met with in people of very respectable surroundings. The irritation caused by the parasites and the rubbing induced by itching produce a congestion of the lid-margin, while the pediculi with their excreta and their numerous eggs clinging to the roots of the cilia bear a close resemblance to the crust seen in blepharitis marginalis, for which this affection may readily be, and doubtless often is, mistaken. This is the more likely to happen, as the mercurial preparations prescribed for the blepharitis destroy the pediculi. Phthiriasis ciliorum is probably not so rare as it is generally stated to be. Schwenk[2] found 19 cases in 19,819 consecutive patients at the dispensaries of the Wills' and Pennsylvania Hospitals.

The pediculi pubis sometimes also infest the eyebrows, but the pediculi capitis are never found in the lashes or brows, even when they abound on the patient's head.

Thorough cleansing with absorbent cotton wet with a solution of bichloride, 1 to 4000, and the subsequent application of yellow oxide or other mercurial ointment, will soon destroy the parasites.

Trichiasis and **Distichiasis.**—The distinction between these two terms has little practical importance. The essential condition in each is

[1] Michel, Graefe u. Saemisch, Bd. iv. S. 411.
[2] Wills' Eye Hospital Reports, vol. i.

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the abnormal direction of the lashes, which are turned in upon the ball and become a source of irritation instead of a protection. Trichiasis is properly applied only to cases in which the deviation in the direction of the lashes is not accompanied by inversion of the margin of the lid; in other words, when this deviation is an independent affection and not merely part of the condition found in entropion. Its usual cause is prolonged inflammation of the lid-margins; when the distortion of the lashes is a result of more general disease of the conjunctiva and cartilage, the edge of the lid is not likely to be found in its normal position. It is not always easy to decide where trichiasis ends and entropion begins, and it is hardly possible to discuss them separately without some confusion. Either inflammatory swelling or secondary cicatricial contraction of proliferated connective tissue may readily displace the orifices of exit of the lashes and give the latter a vicious direction, while inflammation extending to the follicles may cause an irregular growth. The margin of the lid is usually thickened, and the posterior free edge loses its definite outline. Cilia may be deviated from their normal direction congenitally. I have met with several cases in which a lash was deviated anteriorly and in process of growth had forced its way beneath the epithelium of the skin of the lid. The attention of the patient was called to a small bleb near the margin of the upper lid, and when this was opened a fully developed cilium of normal thickness and color was found. In a lady of middle age, who has never suffered from blepharitis, I have removed such a lash three times from the same position in the course of several years.

Distichiasis is a term that has been rather loosely used. Strictly speaking, it indicates that there are two distinct rows of lashes, the posterior row being considered supernumerary; but it is quite generally applied to cases in which there is an excessive number of hairs, some of which grow in abnormal positions and turn in upon the ball. The cilia normally form several rows, which are placed so nearly in the same plane as to present the appearance of being ranged in a single line, and their number naturally varies greatly in different persons. Some authors (Scarpa, Mackenzie, Wecker, and others) deny the existence of distichiasis, and maintain that the condition so called is merely the result of an abnormal direction of some of the hairs, and not of an increase in their number. Other authorities (Wilde, Michel, Raehlmann, and others) insist upon an additional growth of hairs, and the older authors particularly speak of "preternatural hairs," "pseudo-cilia," and "supernumerary lashes." Carter objects to the distinction between "trichiasis" and "distichiasis," but has "frequently seen a growth of cilia in abnormal numbers, some of them being misdirected." Stellwag says of distichiasis that "the condition occurs very rarely, and is then generally congenital. The pseudo-cilia are developed either in children or at the time of puberty, when the growth of hair on other parts of the body is accelerated. It occurs more rarely in the later periods of life. In by far the greater number of cases the double growth is only apparent."

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Michel says that an increased number of cilia may be either congenital or acquired. In congenital distichiasis two, three, or four rows of cilia have been observed, and they may occupy a part or the whole of the lid-margin. In acquired distichiasis, resulting from long-standing inflammation of the hair-follicles, the extra lashes are crowded and bunched together without regularity. Two cilia may grow out of one follicle with divergent direction. Increased size of lashes may result from inflammatory action, as may also atrophy of bulbs, after which fine soft hairs will frequently take an abnormal direction.[1] It seems to be generally admitted that cases of congenital distichiasis, monolateral or bilateral, partial or complete, are occasionally met with. They are, however, so very rare as to have comparatively little interest for the practical surgeon. If by distichiasis we mean an additional row of lashes distinct from the normal one, it is very questionable if any such condition can be produced by disease; but whether additional cilia or, at all events, a new growth of hair on the margin of the lid may be a result of long-continued inflammation is another question. Unna[2] states that he has quite frequently seen sprouts, projecting laterally from the sheaths of the lashes and developing like new sheaths, which follow an entirely different direction in the tissue of the eyelid, and that from these are developed hairs which take an abnormal course. Raehlmann[3] says that new hairs may appear as offshoots from the follicles of the cilia, and also by primary development from the cuticle of the free margin of the lid. Hairs far removed from true cilia, which may or may not be growing in a normal direction, he believes are developed from the epithelial covering of the lid-margin,—a post-fœtal development of hair from the epidermis. This raises an interesting question about which authorities differ. Stricker refers to it only briefly and incidentally, as follows: "Hairs shed in consequence of disease are either not succeeded by new hair, or in their place lanuginous hairs are formed." It would seem that these lanuginous hairs, whose follicles are situated in the superficial part of the corium, if found upon the lid-margin can have no connection with the bulbs of the cilia, but must have their origin as described by Raehlmann, and that the fine colorless hairs that often grow from the free lid-margin and give so much trouble to patient and surgeon are not, properly speaking, lashes. It seems probable that a pathological increase in the number of hairs on the lid-margin may be only apparent, from a separation of the points of exit of some of the true cilia by inflammatory thickening of the margin, or may be a result of the development of offshoots from the bulbs of the cilia, or of the new formation of superficial hairs developed from the epithelium.

Entropion.—The free margin of the lid is inverted, turning the lashes against the ball; the lashes may be normal in reference to the lid, or may

[1] Graefe und Saemisch, Bd. iv. S. 410.
[2] Arch. für mikrosk. Anatomie, Bd. xii., 1876.
[3] Graefe's Archiv, Bd. xxxvii. S. 2.

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be themselves displaced in position and distorted in form, complicating the entropion with trichiasis. There are two forms of entropion, which may be more or less distinct, the *spasmodic* and the *cicatricial.* Some authors add a third, giving the name of "bulbar entropion" to cases depending upon loss of the support which the lids normally receive from the ball, in consequence of retraction of the latter within the orbit or of its atrophy or absence; but this seems rather an unnecessary refinement in nomenclature.

In the spasmodic form, the *acute entropion* of Mackenzie and other authors, the cartilage may be in normal condition and the lid-margins may be healthy but simply incurved by spasmodic contraction of the ciliary fibres of the orbicularis. This occurs usually, if not universally, in the lower lid, whose thin narrow and more pliant cartilage and its position against only the lower part of the anterior convexity of the ball furnish mechanical conditions favorable to this displacement. The predisposing causes of this form of entropion are redundancy or relaxation of the skin of the lid and loss of the support normally furnished by the eyeball. Redundancy of the skin is occasionally met with in young children as a congenital condition; the other predisposing causes are usually incident to old age, and result from the disappearance of fat from the skin and orbit. The effect produced by recession of the ball may, however, follow phthisis bulbi from any cause, and the lashes often give trouble by turning in upon the conjunctiva if an artificial eye is not worn after enucleation. The exciting causes are conjunctival and corneal irritation and intense photophobia, which excite reflex contraction of the orbicularis. When once induced, the entropion is maintained and intensified by the irritation that it produces. In the phlyctenular ophthalmia of children, the effect of muscular spasm is aided by inflammatory swelling of the skin of the lid. Another exciting cause, and one which must be guarded against in the after-treatment of cataract operations in aged subjects, is prolonged bandaging. When the inversion is complete, the lashes are buried out of sight in the conjunctival fold, where they do less mischief than when they are in contact with the cornea. In uncomplicated spasmodic entropion the margin of the lid readily resumes its normal position when slight downward traction is made with the finger on the skin, but turns in again with a jerk immediately or in a few minutes after the finger is removed.

Chronic or cicatricial entropion is a different and much more serious condition. Muscular spasm may aid in its production, but its chief causes are contraction of the conjunctiva and distortion of the cartilage. As it is the result of long-continued inflammatory processes, the bulbs of the cilia are not likely to escape involvement, and it is usually accompanied by trichiasis. It is easy to understand how the natural curve of the tarsus, adapting itself to the convexity of the ball, may be increased by cicatricial contraction of the conjunctiva and of the surface of the cartilage lying next to it. There is usually more or less horizontal, as well as vertical, contraction, which narrows the commissure, producing the condition known as

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blepharophimosis, and increases the tendency to inversion. In very high degrees of conjunctival atrophy the retrotarsal fold may be nearly lost, while vertical folds appear which produce the impression that the ball is adherent to the posterior part of the lid, and constitute the "symblepharon posterius" of Von Ammon. The causes that produce spasmodic entropion may aid in the production of chronic inversion. The traumatic form of entropion is the result of cicatricial contraction following injuries of the conjunctiva and subconjunctival tissue by burns, or by the application of acids, alkalies, or other destructive agents.

The most frequent cause of chronic entropion is trachoma. The exudations which in this obstinate and chronic disease take place in the palpebral conjunctiva and subconjunctival tissue result in cicatricial contraction and produce the condition, marked by a smooth glistening surface with whitish lines of cicatrix more or less distinct, which is so painfully familiar to all ophthalmic surgeons. The most constant and decided of these lines is found two or three millimetres above the lid-margin, and its production is explained by Arlt[1] as follows. He says that in cases of long-standing trachoma, deep infiltration takes place, especially in the line corresponding to the entrance of the palpebral arteries next to the free border of the lid, resulting in exudations which, embedded in the form of granules or nodules, displace the tissue in which they are deposited. By this supplanting of the tarsal tissue, which for the most part takes place along the above-mentioned line, and by the subsequent contraction, the tarsus is incurved. The narrow border between the edge of the lid and this cicatricial strip is diminished by contraction, the sharp inner edge of the lid-margin is obliterated, and the cilia are turned against the globe.

Congenital entropion of the lower lid is comparatively not very uncommon. It is usually due chiefly to a redundancy of the skin. According to Michel, there is an hypertrophy of the ciliary portion of the orbicularis muscle. Congenital entropion of the upper lid is extremely rare. A case reported by Von Ammon[2] is sometimes quoted as unique. He describes and figures a case of congenital entropion of both lids in the left eye, and of the upper lid in the right. In the latter there was also ectropion of the lower lid. Wilde[3] reports a case of congenital entropion of the upper lid.

Ectropion, or eversion of the margin of the eyelid, is produced by various causes. Acute or spasmodic ectropion is the result of swelling of the conjunctiva and spasm of the orbicularis occurring in acute inflammation of the conjunctiva, usually of the blennorrhœal form. It is sometimes met with in the phlyctenular conjunctivitis of children, in whom crying and violent resistance to examination of the eyes excite orbicular spasm.

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[1] Diseases of the Eye, Amer. ed., p. 39.

[2] Klinische Darstellungen der angeborenen Krankheiten des Auges und der Augenlieder, S. 6.

[3] Dublin Journ. of Med. Sci., xxv.

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The œdema of the skin subsides, while the chemosis continues, the bulging conjunctiva puts the ciliary portion of the orbicularis on the stretch, and contraction of the orbital fibres everts the lid. The retroverted lid and contracting orbicularis act like a ligature, and tend to keep up the condition by strangulating the conjunctival veins. The lids can be replaced temporarily by the fingers, and usually resume their normal position as the inflammation subsides. The exposed conjunctiva may, however, become permanently hypertrophied, assuming a fleshy appearance and causing the form of ectropion which has been called *sarcomatous*. A permanent ectropion may be produced by orbital tumors causing bulging of the conjunctival fold by pressure upon the veins behind the ball. Exophthalmos also tends to produce eversion of the lid. Paralytic ectropion may vary from a slight displacement of the lacrymal punctum, from paralysis of Horner's muscle, causing a troublesome stillicidium, to decided eversion of the lower lid in lagophthalmos. Much the same condition is found in senile ectropion, in which the muscle is atrophied and the relaxed and flabby lower lid falls away from the ball by its own weight. Ciliary blepharitis, resulting in hypertrophy of the lid-margin and distortion of the cartilage, is a frequent cause of ectropion. Eversion of the lower lid, however it may originate, is promoted by the contraction of the skin produced by excoriation from lacrymal and conjunctival discharges. Obstruction of the tear-passages may act in this way as the essential cause.

In cicatricial ectropion the margin of the lid is dragged outward by the contraction of cicatrices in the neighboring skin resulting from injury or local disease. Burns are the most common cause. Abscess of the skin followed by considerable loss of substance, or of the orbital tissue, is an occasional cause, as is also the cicatrization of syphilitic or epithelial ulcers. A very troublesome form of ectropion is produced by cicatrices adherent to the bone, as in cases of fistula of the frontal sinus or of caries of the orbital margin. Lacerated wounds of the lids, particularly if they are not coaptated skilfully or if sloughing takes place, are likely to result in eversion. Ectropion has been met with in a few cases as a congenital condition.[1] The everted lid becomes more or less elongated by relaxation, stretching, or inflammatory hypertrophy. This elongation sometimes proceeds to an enormous extent, particularly in cases resulting from the contraction following burns, when the lid-margin may be drawn far down upon the cheek. The everted puncta cease to carry off the tears, and, in the case of the lower lid, the conjunctival cul-de-sac is obliterated and the tears and conjunctival secretions flow directly upon the skin, producing an excoriation which assists in maintaining and increasing the deformity. The exposed conjunctiva is irritable and congested, and often hypertrophied in the earlier stages; while in long-standing cases the epithelium becomes indurated and

[1] Von Ammon, Klinische Darstellungen der angeborenen Krankheiten des Auges und der Augenlieder, S. 1, Pl. 1, Fig. 7.

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the membrane is dry and shrivelled. The cornea is usually to a great extent protected by forced rotation upward of the ball, even in ectropion of the upper lid when the orbital margin of the tarsus is pressed downward by contraction of the orbicularis, but is liable to suffer from repeated attacks of keratitis which may result in a condition of pannus or even in ulceration or destructive sloughing.

Blepharophimosis means a contraction of the commissure. It may be the result of cicatricial contraction of the lids following trachoma, or may be produced by adhesion of the lid-margins at the external canthus due to the baring of the opposing surfaces by ulceration or long-standing inflammation.

Ankyloblepharon is an adhesion of the margins of the lids not confined to the canthus. It is liable to occur when the opposing lid-margins are bared from any cause, but is most frequently the result of burns. Extensive ankyloblepharon uncomplicated with symblepharon is rare. When the latter does not exist, the eyeball can be moved freely without dragging upon the lid.

Symblepharon is an adhesion of the lid to the ball, resulting from the destruction of the conjunctival epithelium. Burns from powder or chemicals or molten metal are the most frequent cause. It is rare as the result of conjunctivitis, except of the diphtheritic form. In symblepharon anterius the lid-margin is adherent but the cul-de-sac remains free, and a probe can be passed along it behind the adhesions. In total symblepharon the whole lid is adherent and the cul-de-sac is obliterated. Partial symblepharon may involve the cul-de-sac to a limited extent. The adhesions in some cases are stretched into bands by the movements of the ball. A cicatricial contraction of the fornix following severe trachoma sometimes restricts the motility of the eye, and has been called symblepharon posterius. The treatment of these conditions is entirely operative. (See article on Operations.)

Fissure of the Canthus, though not usually mentioned in the text-books, is a complication that frequently requires attention in the treatment of some forms of ophthalmia. In cases of conjunctivitis and keratitis accompanied with considerable photophobia and orbicular spasm, a little groove is sometimes formed by the continued folding of the skin at the outer canthus which is excoriated by the discharges from the inflamed conjunctiva, and a superficial ulceration is produced. The reflex irritation of this fissure increases the orbicular spasm and becomes an important factor in maintaining the irritable condition of the eye. Slight cases can generally be relieved by touching the part with the point of a mitigated nitrate of silver pencil; when the blepharospasm is decided and obstinate a canthotomy will be useful; while in chronic cases with a tendency to blepharophimosis the more permanent effect of a canthoplasty may be required.

Blepharospasm may be a symptom of a neurosis affecting the facial nerve, but is usually a result of some local or distal reflex irritation. In

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its mildest form there is simply a twitching of a few fibres of the orbicularis, which, though giving some annoyance to the patient, is scarcely noticeable without close inspection. This is met with most frequently in delicate women. Children are often affected with a constant winking which does not depend upon any local cause. It is choreic in character, and may extend to the other muscles of the face, or may even be the first manifestation of general chorea. Blepharospasm occurs occasionally as a symptom of hysteria. Of two cases of this kind occurring in my practice, violent and persistent clonic spasm of the orbicularis was cured in one by the free inhalation of nitrite of amyl, and in the other by mental impression.[1]

Constant and more or less violent nictitation sometimes exists as a permanent condition, lasting for a lifetime without known cause or other symptom of neurosis.

Cases plainly due to reflex irritation of the fifth nerve are those in which the spasm is induced by foreign bodies in the cornea or conjunctiva, iritis, cyclitis, and particularly phlyctenular ophthalmia. The blepharospasm is aggravated in these cases by exposure to light, and is accompanied by photophobia, which, however, cannot, strictly speaking, be considered a cause. Defects in refraction and in muscular balance and nasal irritation are possible causes.

The most violent cases of blepharospasm are those that are met with in connection with trifacial neuralgia (tic douloureux). The patient may be quite blind while the spasm lasts. There are periods of complete remission, and, as in the other forms, there is entire cessation during sleep. In some cases the spasm can be temporarily arrested by pressure on the supra- or infra-orbital nerve, or on other "pressure-points" along some of its branches, or on the facial at the stylo-mastoid foramen. The usual cause is probably some local or distant reflex irritation, the source of which cannot always be discovered but should be diligently searched for. Attention has recently been called to cases originating in suppuration of the sinuses adjacent to the orbit.[2]

When the blepharospasm is a manifestation of chorea, tonics, fresh air, hygienic measures, and other remedies resorted to in this disease are required. Among drugs, arsenic is most likely to be useful. When the spasm is the result of reflex irritation, the cause should, of course, be found and removed if possible. If this cannot be done, treatment is to a great extent empirical and too often only palliative. In all cases existing errors of refraction should be corrected by glasses and any considerable degree of heterophoria remedied by prisms or tenotomy. Accommodative spasm may be relieved by atropia. If there is photophobia, smoked glasses will be useful. A fissure of the canthus may require attention. In a case

[1] Trans. Amer. Ophth. Soc., 1884.

[2] Berger, Maladies des Yeux dans leurs Rapports avec la Path. gén., p. 192.

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uncomplicated with neuralgia and apparently resulting from a blow upon the temple, Mathewson,[1] after various kinds of treatment had failed, effected a cure by opposing the action of the orbicular muscle with the constant tension of a rubber band attached to the lid and to the brow by means of collodion. The neuralgic cases are the most difficult to treat, and the list of anti-neuralgic remedies is usually exhausted in vain. Subcutaneous injections of morphia give great temporary relief and in a few cases have a more permanent result, but great caution should be observed in their use. The continuous current is sometimes useful. A moderate current is used with the negative pole applied to the back of the neck and the positive to the muscle or to the points of compression (Michel, Wecker). Section of the supra- or infra-orbital nerve is generally temporary in its effects, but is a simple operation and may be worthy of a trial. Excision sometimes affords relief, for several months at least. According to some authors (Panas), stretching of the nerve is more efficient than either of these operations.

Sympathetic Spasm of the Eyelids (Graefe symptom) was described by Graefe[2] as a symptom in exophthalmic goitre. When the patient looks directly forward, the upper lid does not reach the cornea, but leaves a space of sclerotic uncovered, and when he looks down, the lid does not follow, or follows only partially, the movements of the ball. The lower lid is slightly depressed. This symptom is not constant in goitre, and is met with more frequently in the earlier stages of the disease. Wecker has observed it also in hysterical and in pregnant women and in cases of locomotor ataxia. It is attributed to spasm of the unstriated muscular fibres of Müller. Remak found that irritation of the cervical sympathetic caused elevation of the upper lid.

Sympathetic Paralysis of the Lid has also been observed, accompanying myosis, in paralysis of the cervical sympathetic.[3] There is slight drooping of the lid without impairment of voluntary motion.

Paralysis of the Orbicularis Palpebrarum (Lagophthalmos) occurs usually in connection with paralysis of the other muscles of the face, though the orbicularis often escapes when the other muscles are involved. The facial nerve may be injured at its point of emergence from the stylo-mastoid foramen, or upon the face, or during its progress through the temporal bone, or may be paralyzed as the result of an intra-cranial lesion. Among the extra-cranial causes are wounds, pressure by enlarged parotid or by periosteal swelling, inflammation of the nerve itself or of its sheath, suppurative otitis, and fracture of the base of the skull. The orbicularis is especially liable to be involved in the paralysis of the muscles of the face occurring in leprosy. Paralysis of the orbicularis from intra-cranial causes is very rare. When met with in connection with hemiplegia it may be found on the same side with it or on the opposite side. Lesions

[1] Trans. Amer. Ophth. Soc., 1874.
[2] Berlin. klin. Wochenschr., No. 31, 1867.
[3] Horner, Klin. Monatsbl. f. Augenheilk., Bd. vii. S. 193.

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in front of the pons or in the anterior portion of it cause facial paralysis on the same side as the hemiplegia; below the pons or on the posterior part of it, on the side of the lesion, and therefore opposite to the hemiplegia. The dividing line is said to be one crossing the pons and passing through the roots of the fifth nerves,—the "line of Gubler."[1] When the orbicularis is paralyzed the patient suffers from epiphora on account of eversion of the puncta, and the cornea and conjunctiva are injured by exposure. The cornea frequently ulcerates if not protected.

In the early stage of the acute cases, such as frequently result from exposure to cold wind or draught, leeches and hot stupes should be applied. Later the continuous current is often very useful. Hypodermic injections of strychnia are recommended. Mercurials and iodides should be administered if there is suspicion of syphilis. The cornea should be protected by closing the lid with plaster or a compress bandage. If the paralysis is permanent, a tarsorrhaphy will be required.

Ptosis, or falling of the upper lid, varies in degree from a failure of the lid to follow the motion of the ball in looking upward to complete closure of the eye. It may be due to a variety of causes, such as increased weight of the lid from inflammatory hypertrophy or morbid accumulation of fat, tonic spasm of the orbicularis from long-continued reflex action in photophobia, traumatic injury of muscle or nerve, imperfect development or defective innervation of muscle, and paralysis.

Paralysis of the levator may be central or peripheral, and is frequently accompanied by paralysis of the other orbital muscles supplied by the third nerve. It is sometimes hysterical. Probably of the latter form are cases of transient ptosis occurring after sleep, in which it is impossible to open the eye until the lid has first been raised by the fingers.[2] The slight drooping, noticed only when the eye looks upward and the lid fails to follow the motion of the ball, has been attributed to paralysis of Müller's muscle. It is associated with slight elevation of the lower lid, contraction of the pupil, hypersecretion of the conjunctiva, increased temperature of the side of the face, and diminished tension of the ball, and is a symptom of disease or injury of the cervical sympathetic.

Congenital ptosis is met with in several forms. The hypertrophic, or that dependent upon excess of the skin of the lid, is not frequent. It occurs sometimes in connection with epicanthus. The atrophic form is described as being due to defective development or absence of the levator muscle, and is sometimes hereditary; it cannot always be distinguished from the paralytic. The latter is thought by most authorities to be the result, in a large proportion of congenital cases, if not in all, of injury inflicted by excessive pressure upon the cranium during delivery.

Treatment.—Paralytic ptosis may sometimes disappear spontaneously, or

[1] Ranney, Lectures on Nervous Diseases, p. 74

[2] Harlan, Annales d'Oculistique, January, 1877, p. 83.

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be successfully treated without operation. If of syphilitic origin, which is the most frequent, it will often yield to mercurials and iodides. The rheumatic form is not very uncommon, and is amenable to appropriate treatment. Galvanism is often useful in the later stages. Subcutaneous injections of strychnia are recommended. Several months should be allowed to pass before operation is resorted to. (See article on Operations.) If one eye only is affected and one or more of the muscles of the ball are also paralyzed, the ptosis may be a less evil than the annoying diplopia that would occur if the eye were opened.

Congenital Malformations and Abnormalities, and Wounds and Injuries, of the Lids are discussed in special articles. The surgical treatment of the former and of the results of the latter is described in the article on Operations upon the Lids. The treatment of recent wounds of the lids is favored by the vascularity of the parts. Horizontal wounds usually need only careful stitching with fine thread, unless the tendon of the levator is separated from the cartilage, when an attempt should be made to reunite it. This was done with success by Green[1] two years after the occurrence of the injury. Wounds involving the lid-margin require great care in apposition to prevent deformity after union. The line of the cilia should be carefully preserved. A fine needle and a harelip suture at the edge of the lid will sometimes be useful. Sutures may be required in the conjunctival surface to maintain the edges of the divided cartilage in position. Only very fine stitches should be used. The permeability of the lacrymal passages should be preserved if possible. This may sometimes be done, before cicatrization has taken place, by slitting up the remains of the canaliculus and passing probes into the sac. A compress bandage promotes rapid union by securing immobility. Iodoform or aristol dusted over the surface of the wound, or lint wet with bichloride 1 to 5000 placed upon it, makes a good antiseptic dressing. Iced compresses may be required if there has been extensive laceration.

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[1] Trans. Amer. Ophth. Soc., 1871, p. 134.

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OPERATIONS PERFORMED UPON THE EYELIDS.

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OPERATION FOR CHALAZION.

CHALAZIA may be removed through either the skin or the conjunctiva. If the cyst is of large size and nearer to the skin, it may be better to operate externally than to make an extensive incision of the conjunctiva and cartilage and run the risk of serious subsequent contraction. When this is done the operation is sufficiently painful to justify etherization. A Desmarres ring forceps or Snellen clamp is used to give complete control of the lid and prevent bleeding. A horizontal incision is made through the skin and the muscular fibres are separated to lay bare the sac, which is grasped and drawn forward by forceps and dissected out with the points of a small pair of scissors curved on the flat. If the skin has been much distended, a small semilunar or oval piece of it may be removed. The cartilage and conjunctiva should be spared as far as possible. The wound is closed by two or three very fine sutures, which are to be removed as soon as the edges have united, generally in twenty-four hours. No special dressing is required; but iced cloths will diminish extravasation and œdema of the lid and promote the comfort of the patient.

Usually the incision is made through the conjunctiva after thorough cocainization. The clamp, which is more painful than the knife, may be dispensed with and the lid be everted on the end of a horn spatula while the cyst is cut open. The gelatinous contents are then scooped out with a sharp curette, with which also the walls of the sac are scraped sufficiently to excite adhesive inflammation. The latter object is sometimes accomplished by means of cauterization with the mitigated nitrate of silver stick; or a part of the cyst-wall may be seized with the forceps and dissected out.

OPERATIONS FOR TRICHIASIS AND DISTICHIASIS.

Operations whose object is to produce an eversion of the whole lid-margin or to transplant the bulbs of the cilia will be considered in discussing the treatment of entropion, with which cases of trichiasis demanding such procedures are always associated. Surgical treatment of simple

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trichiasis is directed to repeated removal of the offending lashes, correction of their deviated positions, or destruction or excision of their bulbs. Simple extraction of an inverted lash by means of the cilia forceps can scarcely be considered more than a palliative measure, though in rare cases after many repetitions of the process the bulb seems to atrophy and the lash ceases to grow. When only one or two lashes are involved and the rest of the lid-margin is in normal condition, it is often good practice to be content with the relief obtained in this way rather than to risk impairing the integrity of neighboring hair-bulbs by more radical operations. Patients themselves, or their friends, may sometimes be taught to perform this little operation and be intrusted with the forceps; but this requires caution, as they are likely to prove heroic practitioners and to attack healthy cilia. It is not uncommon to find patients attributing their discomfort to the tangible cause of "wild hairs" and freely pulling out innocent lashes when no trichiasis has existed. A troublesome and obstinate irritation of the eye, sometimes accompanied by a small corneal ulcer, may be caused by a minute colorless inverted lash that might easily escape detection unless carefully sought for by oblique illumination and a convex lens. In circumscribed trichiasis, involving a small group of lashes, some surgeons (Wecker) think well of the Gaillard suture. A thread is entered through the skin at the edge of the lid near the deviated lashes, passed deeply underneath the muscle, brought out eight millimetres above the lid-margin, and firmly tied. It is left to slough out, and forms a cicatricial band which maintains the lashes everted. Attempts have been made to give a proper direction to individual inverted lashes, but not with very satisfactory results. Celsus is not responsible for the proceeding (*illaqueatio ciliorum*) to which his name is sometimes given, but to which he merely refers and without approval. "Some allege that 'tis proper to pierce the external part of the eyelid near the eyelashes with a needle, which must be passed thro' with a woman's hair doubled for a thread; and when the needle has gone thro', that the offending hair must be taken up into the loup of the woman's hair, and by that drawn upward to the superior part of the eyelid, and then to be glued down to the flesh, and a medicine applied to close up the orifice thus made."[1] He gives several reasons for considering this method of disposing of "preternatural hairs" impracticable. Snellen[2] described practically the same proceeding, except that a thread was substituted for the woman's hair; Knapp passed the lash through the eye of the needle instead of through the loop; and Wecker invented a needle with a little hook instead of an eye for catching the lash, but finally came to the conclusion which Celsus reached, that such proceedings are impracticable, particularly in view of the fact that the cilia only live three or four months and their successors cannot be depended upon to follow the correct path. Argyll Robertson advocated this operation and

[1] Greive's Celsus, p. 399.

[2] Wien. Med. Wochenschr., xxiii., 1871.

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thinks a successful result is usually permanent, although he has not been able to satisfy himself of the process by which it is attained.[1]

Numerous methods of getting rid permanently of the offending lashes have been adopted from time to time. The ancient Egyptians, who from the remotest antiquity had exceptional opportunities for studying diseases of the eye and among whom the first specialists appear in history, destroyed the bulbs and the margins of the lids by cauterization with red-hot plates of gold, and are said to have performed this operation upon the healthy lids of children as a prophylactic measure. They also everted the lashes by producing cicatrices in the skin of the lid by actual cautery or chemical caustics. Heister destroyed the lid-margin with caustic potash, and Rhazes, Saunders, Bartisch, and others cut it away with a knife. Vacca[2] removed a strip of skin a line and a half in width just above the displaced lashes and dissected out the bulbs or destroyed them with nitric acid, without disturbing the cartilage, then replaced the skin and retained it with plaster. Flarer[3] split the lid-margin by an incision made just in front of the cartilage and parallel to its surface, and exsected the bulbs of the lashes with a zone of skin and muscle. This is the operation now sometimes performed under the name of "scalping." It has been condemned as barbarous, but cases are occasionally met with in which it is not only justifiable but may be the best, or even the only, means of giving permanent relief. When the lashes, which in health serve so perfectly the purposes of beauty and protection, have been reduced by disease, assisted perhaps by surgical efforts, to a scant row of irregular, distorted, stubby hairs that cease to be either useful or ornamental, it is not bad surgery to sacrifice them if necessary; and it may become so if some of them emerge from the posterior angle of the lid-margin and grow so directly backward that it seems hopeless to attempt to give them a proper direction by any operation. The edge of the lid is split, as in Arlt's operation (see page 97), and an incision parallel to the lid-margin and about two millimetres above it is made through the skin and muscle only. This separates a narrow band of tissue containing the bulbs of the cilia, which is dissected away. The cut edges of the skin and conjunctiva are then united by sutures. Care must be taken that no bulbs are left to reproduce the trouble. They can be seen as black specks when the exposed surface is cleared of blood. It is well to search for them with a convex lens. If the skin of the lid is insufficient and even this narrow strip cannot be spared, a flap may be turned back from the extreme lid-margin and be replaced after the bulbs have been dissected out.

Various attempts have been made, with more or less success, to get rid of individual lashes by destroying their bulbs. One plan is to insert into the follicle, after the removal of the lash, a platinum needle that has been

[1] Edinburgh Med. Journ., May, 1874.

[2] Annali universali di Medicina, compilati dal dottore Annibale Omodei, 1825.

[3] Zanerini dissert. sopra Trichiasi, Pavia, 1829.

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dipped in caustic potash.[1] It has been objected to this that it seems scarcely possible that an appreciable amount of the fluid could be carried to the desired point in this way, as it must be wiped off in the passage of the needle through the tissue. There is good authority, however, for its success in some cases, and it is possible that even the mechanical action of the point of the needle, if it happen to reach the papilla, might excite sufficient destructive inflammation to prevent the growth of the lash. Steel needles dipped in fused nitrate of silver have also been used.[2] Celsus[3] said that the best means of treating inverted cilia was to destroy their roots by inserting into them a red-hot thin and broad needle; and Ambrose Paré invented a needle fixed in a ball of steel for this purpose. Carron du Villards[4] practised galvano-puncture in 1837.

Dr. Charles E. Michel[5] introduced the method of destroying the bulbs by means of electrolysis. A constant battery of from eight to twenty elements is used. A gilt sewing-needle (No. 8) connected with the negative pole is pushed into the follicle by the side of the lash, and the sponge connected with the positive pole is placed on the patient's temple or held in his hand. When the circuit is closed, a slight frothing, from the escape of minute bubbles of gas, is seen around the stem of the needle, which is the sign for breaking the current. With eight ordinary-sized elements the desired effect is produced in from two to five seconds; with a stronger current less time is required. The lash comes away with the needle or is withdrawn by very gentle traction, if the application has been successful; otherwise the papilla has not been reached or the decomposition has not been sufficient and the operation should be repeated. This method often answers well when only two or three lashes are inverted, but is rather painful and tedious when more are to be disposed of. It can, of course, be done at several sittings, or an anæsthetic may be used. The reaction is generally slight. When the hair is very fine, or irregularly placed it is a difficult matter to strike its bulb accurately with the needle. Dr. Michel, however, claims that absolute contact is not necessary. This method seems rational, and has perhaps hardly had so fair and extensive a trial as it deserves. I have used it in a number of cases at the Wills' Hospital with some degree of success, which might probably be improved by greater technical skill acquired in longer and more patient practice.

OPERATIONS FOR ENTROPION.

If it be true that the curability of a disease is in inverse proportion to the number of remedies proposed for its cure, a glance at the rather extensive literature of entropion will at once suggest that it is a very difficult

[1] H. W. Williams, O. H. R., vol. iii. p. 219.
[2] Michel, St. Louis Clinical Record, October, 1875.
[3] Greive's Celsus, p. 399.
[4] Maladies des Yeux, t. i. p. 307.
[5] St Louis Courier of Medicine, February, 1879.

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condition to treat. Beginning with Celsus, there have been few surgeons prominently connected with operations upon the eye who have not either invented an operation for entropion or modified somebody else's. To recount all their procedures would be tedious and profitless, and those only will be described which illustrate the principles upon which different plans of operation are based, or which seem to be in accordance with modern methods.

The various methods devised for correcting the faulty position of the ciliary margin of the lid may be classed as:

I. Those that propose to evert the margin by the removal of a portion of the skin of the lid, or by the contraction resulting from the use of caustics, cauteries, or sutures.

II. Those that depend upon the tension of the skin caused by uniting it with the orbital margin of the tarsal cartilage.

III. Those based upon transplantation of the bulbs of the cilia.

IV. Those in which the tarsal cartilage is incised, or grooved upon its outer or its inner surface.

Entropion of the lower lid dependent chiefly upon redundancy of the skin may be treated rationally and successfully by a judicious removal of a portion of the latter. A horizontal oval flap, the size of which is determined by the amount of excess of skin and the extent of the entropion, is taken from the lid near its margin, and the edges of the wound are brought together by stitches. A fold of skin may be raised by one of the numerous forms of "entropion forceps," transfixed by a Graefe or Beer cataract knife, and cut out close to the blades of the forceps; or the lid may be held in a Snellen clamp while the flap is outlined with a knife and dissected away with a pair of small scissors. As has been already shown, this condition of the skin is met with usually in old age and rarely in the congenital form of entropion. I have seen only two cases of the latter. One was easily cured by the scientific removal of a flap of skin; in the case of the other I was cheated of my operation by a large dog that bounded upon the child and scratched a wound in its lower lid, but the result was equally satisfactory.

In the spasmodic form, the "acute entropion" of Mackenzie, in the production of which irritative contraction of the marginal fibres of the orbicularis is the chief factor, more or less fulness or relaxation of the skin is usually a predisposing cause, and here also a partial removal of it is often the best procedure. Vertical flaps of skin have sometimes been excised (Celsus, Janson, Graefe), and may be advisable in exceptional cases; but they are liable to the objection that a permanent scar is left, while a horizontal cicatrix falls in with the natural folds and soon becomes invisible. When there is decided spasm of the orbicularis the excision of a strip of muscular fibres will add to the effect of these operations; and subcutaneous myotomy of the whole muscle, from the orbital border to the free margin of the lid, has been recommended by Dieffenbach, Cunier, and Heidenreich.

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Some surgeons think well of the Gaillard sutures described under Trichiasis; and the formation of cicatrices by actual or potential cautery was formerly practised, first by the Egyptians, but may be considered obsolete.

When the cause is removable and the condition therefore transient, temporary relief may be afforded by simpler means. If the skin of the lid is well dried and painted with collodion, the contraction which follows evaporation will in some cases be sufficient to hold the lashes away from the eyeball. The collodion will be more efficient if applied over light gauze ("Donna Maria") or thin strands of absorbent cotton. Strips of rubber plaster, which resists moisture better than any other, applied with one end at the edge of the lid and the other extending down to the cheek, will often answer the purpose well. If these means fail, a fold of skin may be raised by the fingers or forceps and two sutures be passed through its base; or an expedient suggested by Graefe[1] may be resorted to. A thread is passed through a little fold of skin near the ciliary border and tied, and one end is cut off near the knot; a similar thread is placed near the margin of the orbit, and the two are tied together over a roll of chamois-skin.

When the entropion is the result of chronic inflammation, and is due to contraction of the conjunctiva and distortion of the tarsal cartilage, none of these measures will be found permanently effective. If more attention had been paid to the statement made by Mackenzie, forty years ago, that "in chronic inversion we generally find that nothing done to the skin merely is of much service," the progress to a rational treatment would have been more rapid and much bad surgery would have been avoided. Within my own recollection, the treatment of all forms of entropion by removing more or less of the skin of the lid was quite general, and that most melancholy picture of hopeless distress, a patient in whom the unoffending skin had been pared away by repeated operations until it was no longer possible to close the eyes completely, while the contracted conjunctiva and distorted cartilage still held the lashes against the cornea, was not very uncommon.

Anagnostakis,[2] recognizing the inefficiency of a mere removal of a portion of the skin, and particularly the fact that the effect was necessarily to a great extent lost when the lid was elevated, conceived the idea of uniting the skin to the orbital margin of the tarsal cartilage,—a firm point of attachment which would keep up the tension in all positions of the lid. Further, tension made from this point acts to great mechanical advantage in everting the free margin of the lid, as can be easily seen by pushing back the skin with a pencil or probe placed in this position. An incision is made parallel to the margin of the lid and about three millimetres above it, passing through the skin only. While the assistant draws the upper edge of the wound well back by stretching the skin of the lid, a band of

[1] Heidelberg Congress, 1868.

[2] Annales d'Oculistique, July, 1857.

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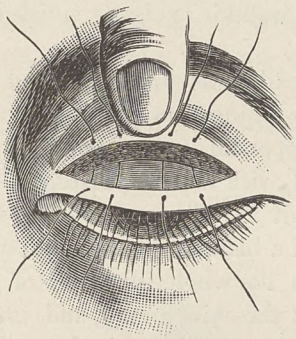
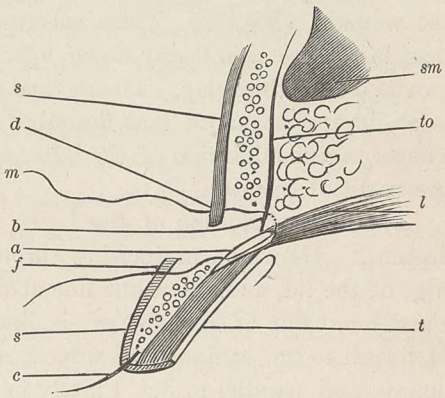
the muscular fibres of the orbicularis, corresponding in length to the extent of the incision, is dissected out over the upper part of the tarsal cartilage. Three or four sutures are then passed through the lower edge of the cutaneous wound and inserted deeply in the fibro-cellular tissue which covers the part of the cartilage from which the muscle has been removed. (Fig. 1.) When these sutures are tied, the narrow strip of skin above the edge of the lid is united to the upper margin of the tarsal cartilage, and is held there permanently by a dense cicatrix. The sutures are allowed to remain until they slough out. The upper edge of the cutaneous wound is allowed to unite without stitching.

Fig. 1.



After Anagnostakis.

Fig. 2.



Vertical section of eyelid.—*sm*, supraorbital margin; *to*, fascia tarso-orbitalis; *l*, levator tendon; *t*, tarsus; *m*, orbicularis muscle; *c*, eyelash; *s*, skin; *f*, lower border, *d*, upper border, of wound; *a*, *b*, passage of suture through aponeurosis. (After Hotz.)

Though this operation was founded on rational principles, and was a great advance over any other that had been practised or proposed, it does not seem to have been adopted, and was entirely forgotten until revived by Dr. Hotz, of Chicago, who, in 1879,[1] without knowledge of Anagnostakis's suggestions, published an operation identical with his in principle, though differing somewhat in detail. He makes the first incision higher up, along the upper margin of the cartilage, extending from one commissure to the other. He defines the upper border of the cartilage by the furrow in the skin beginning two millimetres over the inner canthus, ascending gradually to the middle of the lid, and thence descending to a point two millimetres above the external commissure, and claims that an incision made exactly in this furrow gives the best cosmetic effect, the most natural movement of the lid, and the most complete eversion of the cilia. Neither lid-clamp nor horn spatula is used. While an assistant fixes the skin of the eyebrow against the supraorbital margin, the operator seizes the middle of the free edge of the lid and draws it downward until the convex furrow becomes a straight horizontal line. A horizontal incision is then made through the skin, com-

[1] Archives of Ophthalmology, vol. viii. p. 249.

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mencing two millimetres over the internal canthus and ending two millimetres over the external. This incision is converted into a gaping wound by the retraction of the skin, and a strip of muscle three millimetres in width is removed along its upper margin, exposing the superior border of the cartilage. Four sutures are passed through the lower lip of the wound, the fibrous tissue covering the upper third of the tarsus, the tarso-orbital fascia at its junction with the cartilage, and the skin at the upper edge of the wound. (Fig. 2.) These sutures are tied tightly, so as to draw the margins of the skin firmly down upon the cartilage. The stitches are removed on the third day. The method of performing the "Hotz operation" is an improvement on that described by Anagnostakis, but it should be known as a modification of the latter, as Anagnostakis is clearly entitled to the credit of priority.

The transplantation of the bulbs of the cilia was first suggested by Jaesche.[1] He made an incision through the conjunctiva parallel to the edge of the lid, and about one line above it (upper lid). He then removed a semilunar flap of skin five or six lines broad, with its base corresponding in length to the conjunctival wound, and about two lines above the lid-margin and parallel to it. Finally he entered a bistoury at the middle of the conjunctival incision and passed it through the whole thickness of the lid, causing it to emerge at the lower margin of the skin-wound, and, introducing a pair of scissors into the puncture thus made, separated the lid-margin completely except at its two extremities. This formed a narrow bridge containing the bulbs of the cilia, which, when the edges of the external wound were stitched together, was drawn upward away from the ball and made to occupy the position from which the skin-flap was removed. The suturing of the skin at the same time caused a rotation of the freed lid-margin on its long axis, which turned the lashes outward. His younger brother shows the difference in principle between this operation and that of Arlt, with which he claims that it has been confused.[2]

In Arlt's operation[3] the edge of the lid is split into two layers, and the anterior layer only, containing the cilia, is displaced, the posterior being allowed to remain *in situ*. While an assistant stretches the lid on a horn spatula pressed well up to the conjunctival fold, the operator, with his left thumb placed just above the cilia, draws back the skin so as to evert the margin of the lid and expose the orifices of the Meibomian glands. Then, with a double-edged bistoury slightly bent on the flat or a lance-shaped knife (a Beer cataract-knife answers well) held with one surface looking towards the conjunctiva and the other towards the skin, an incision is made about three millimetres deep, just in front of the Meibomian glands, and extending the whole length of the lid-margin. (Fig. 3.) The lacrymal

[1] Prager Medicinische Wochenschrift, 1845, vol. vii.
[2] Klinische Monatsblätter für Augenheilkunde, 1873, S. 97.
[3] Graefe und Saemisch, vol. iii. p. 447.

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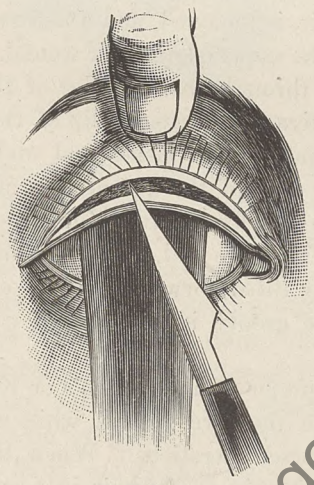
punctum must, of course, be avoided. The anterior lip of this wound will contain the bulbs of the cilia, muscular fibres, and skin, and the posterior lip will be formed of tarsal cartilage, Meibomian glands, and conjunctiva. The next step is to remove a semilunar flap from the skin of the lid. An incision is made through the skin parallel to the border of the lid and three or four millimetres above it, and extending at each end a little beyond the slit in the lid-margin; then, while the assistant fixes the skin on the brow, and the operator, with his index-finger placed just above the incision he has made and the middle finger at the angle of the orbit, uniformly stretches the skin of the lid, the upper curved incision is made meeting the first at its extremities and separated from it three or four millimetres at the middle. The flap of skin thus outlined is dissected out with a pair of straight scissors, and the wound is united with three or four sutures. The stitches are entered near the roots of the lashes, and are made to include the muscular fibres at the lower lip of the wound, but the skin only at the upper.

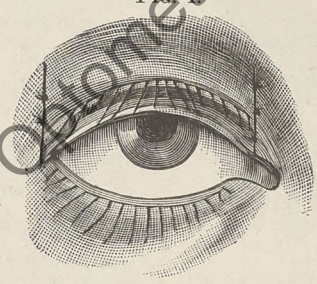
In his earlier operations, Arlt was in the habit of entirely separating the strip of skin containing the lashes except at its extremities, and Wecker still prefers to do so. This makes the operation the same as Jaesche's, except that the anterior lip of the split lid-margin only is transplanted, instead of the whole thickness. It has been objected to this separation of a narrow bridge that the danger of sloughing is incurred; but Wecker thinks that, with antiseptic methods of operating and dressing, this danger may be disregarded.

FIG. 3.



After Arlt.

FIG. 4.



The distinctive principle of Arlt's operation—splitting the lid-margin and sliding back the anterior lamina—is universally admitted to be a good one; the means of retaining the lashes in their new position may be varied with the demands of special cases or the fancy of individual operators. Graefe proposed the following modification.[1] A vertical incision eight or

[1] Archiv für Ophthal., Bd. x., 2, S. 226.

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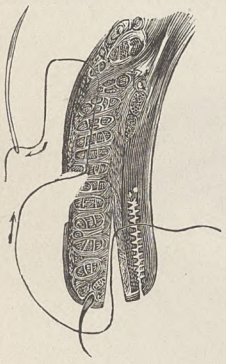
nine millimetres in length is made through the skin and muscle at each extremity of the slit in the lid-margin, and the rectangular flap thus formed is pushed up about four millimetres and held in its new position by sutures at either side. (Fig. 4.) If the skin is abundant, the effect of the operation may be increased by cutting out an oval piece at the upper part of the flap, or by simply pinching up a fold and passing several sutures through its base. This method has the advantages of displacing the lashes near the canthi more completely than is done by the crescentic flap, and of not sacrificing the skin where it is sparse, as the result of previous operations or other cause. Its disadvantage is that vertical incisions are liable to leave permanent scars.

The late Dr. Levis, of Philadelphia, after sliding up the anterior layer of the split margin, held it in place by two or three sutures passed directly through the whole thickness of the lid.

For several years I have operated in such a way that the sutures, while pressing the anterior lip of the split lid-margin upward and tilting the cilia away from the ball, at the same time draw the cartilage and conjunctiva downward. The lid-margin is first split in the usual manner. Then a horizontal strip of skin, three or four millimetres wide and four millimetres above the ciliary margin and parallel to it, is removed, and a narrow band of muscular fibres is dissected out along the upper edge of the wound. The sutures are passed through the free edge of the cartilage and conjunctiva (the posterior lip of the split margin), over the roots of the lashes and under the muscle at the upper edge of the wound, and made to emerge through the skin five or six millimetres above the wound. When they are tied, the strip containing the cilia is strongly tilted forward, and its cut edge is crowded into the groove in the muscle, where it unites. (Fig. 5.)

FIG. 5.



Some surgeons prefer to use Snellen's or Knapp's lid-clamp, instead of the horn spatula, while making the incisions in these operations. When there is marked blepharophimosis, as there is in a considerable proportion of the cases requiring operation for entropion, a free canthoplasty should form part of the procedure.

Among the procedures suggested for transplantation of the cilia may be mentioned one described by Gayet as "une opération autoplastique."[1] The strip of skin taken from the lid, instead of being entirely removed as in Arlt's operation, is left attached at the extremity towards the canthus and transplanted in the groove formed by splitting the lid-margin. The wound in the margin of the lid is made deep enough to gape about four millimetres, and a flap corresponding in size to this wound is formed from the skin of

[1] Annales d'Oculistique, 1882, No. 1, p. 27.

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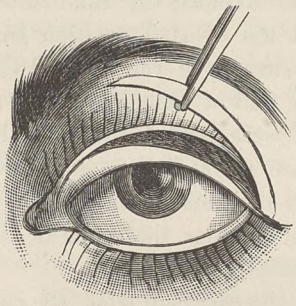
the lid just above the margin and parallel to it. Fig. 6 represents this strip of skin dissected up and held in the forceps ready to be transplanted to its new position, where it is to be secured by two sutures, one at the free extremity and the other at the base.

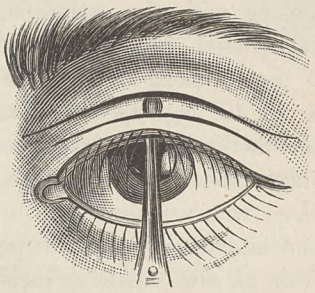
When the entropion is total, and it is necessary to displace the whole range of cilia, Gayet prefers to do two operations at intervals of some days, rather than to transplant at once a long narrow strip of skin, as was his earlier practice. Dianoux,[1] however, gives the preference to the latter plan, and modifies the operation by separating entirely the anterior lip of the split lid-margin except at its extremities. An incision parallel to the margin of the lid and about four millimetres above it, and extending down to the

Fig. 6.



After Gayet.

Fig. 7.



After Swanzy.

cartilage, is made from the region of the lacrymal punctum to the external canthus. A second incision splitting the margin of the lid is made to meet the first, thus completely freeing the bridge of tissue included between them, containing skin, muscular fibres, and the roots of the lashes. A strip of skin about three millimetres wide is now detached from the upper edge of the first wound, drawn down beneath the ciliary bridge, and attached by three sutures to the cut edge of the cartilage. (Fig. 7.) This operation is ingenious, but seems a little dangerous.

Jaesche[2] proposed to modify the Arlt operation by the use of an epidermic flap; and Schröder and Natanson[3] and Gifford[4] have given detailed descriptions of operations including this modification. Jaesche after splitting the lid slid back the anterior layer until two or two and a half lines of the cartilage were exposed, and held it in that position by stitching it to the cartilage. He then covered the exposed surface by a flap of epidermis taken from the arm with a razor. As pointed out by Gifford, the epidermic flaps have an advantage over strips taken from the skin of the lid in containing no hair-bulbs and avoiding the possibility of a growth of lanugo hairs. He considers them preferable to the flaps of Van Millingen, taken

[1] Annales d'Oculistique, 1882, No. 2, p. 132.
[2] Klinische Monatsblätter für Augenheilkunde, 1881, S. 40.
[3] St. Petersburger Medicinische Wochenschrift, 1891, No. 17.
[4] American Journal of Ophthalmology, 1892, No. 1.

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from the mucous membrane of the lip, as they are more easily placed, require no sutures, and adhere more certainly; and he has also transplanted them, with good results, in the gap left by the incision of the conjunctiva and cartilage in the operations of Green and Von Burow. (See below.)

The contracted and incurved cartilage has been attacked in various ways. Guérin,[1] Ware, and others attempted to free it by cutting the whole lid through perpendicularly, producing an artificial coloboma which was allowed to close by granulation. Crampton divided the lid by two perpendicular incisions, one near each canthus, extending from three lines above the orbital margin of the cartilage to the edge of the lid, and not even sparing the canaliculus; the flap thus formed, comprising the whole of the upper lid, was everted against the brow and held in position by a kind of speculum of silver and a bandage.[2] Guthrie[3] avoided the canaliculus, and, dispensing with the speculum, maintained the everted position by three threads passed through the skin near the margin of the lid and fastened upon the forehead by strips of plaster. A fold of skin was also removed from the lid and the edges of the wound were united by these threads. Adams facilitated the eversion by an incision through the conjunctiva and cartilage extending between the two vertical incisions. An incision of the cartilage was recommended by Von Ammon under the name of "longitudinal tarsotomy," and was practised by Richter in 1799.

Von Burow[4] made an incision through the cartilage from the conjunctival side parallel to the edge of the lid and two or three millimetres above it, extending from canthus to canthus. He made a point of the fact that this cut follows the whitish line seen on the conjunctiva of a lid contracted and distorted by trachoma. He then removed a fold of the skin and united the margins of the wound with stitches. According to Himly, Celsus, Aetius, and Paul of Ægina seem to have combined removal of a fold of the skin with a horizontal incision of the conjunctiva only. But reference to the original of Aetius ("Tetrabibli") shows that the conjunctiva is not mentioned, while the context indicates that "subsectionem intrinsecus faciamus" refers to an incision including the cartilage, and that the procedure of this author was substantially the same as that of Von Burow. Aetius also recommended a number of agglutinative compositions for fastening the lashes to the skin of the lid, and sundry applications to prevent them from growing again when they had been pulled out. Among the latter the blood of a frog and the ashes of an incinerated lizard were favorites.

Dr. John Green[5] also makes a longitudinal incision of the cartilage, but accomplishes the eversion of the margin of the lid by means of a peculiar method of suturing. The incision is made through the conjunctiva and

[1] Journal de Montpellier, t. ii. p. 281.
[2] Essay on Entropion, 1806.
[3] Lectures on the Operative Surgery of the Eye, 1825.
[4] Berliner Klinische Wochenschrift, 1873, p. 296.
[5] Trans. Amer. Ophth. Soc., 1880, p. 167.

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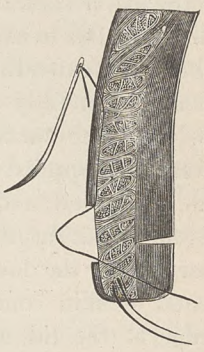
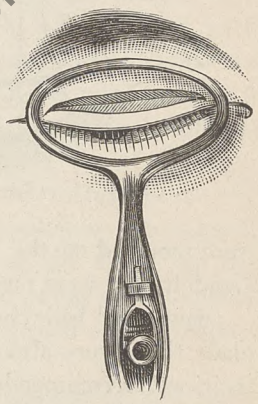
cartilage, parallel to the line of openings of the Meibomian ducts and about two millimetres above them; and a strip of skin, one and a half or two millimetres in width, is excised one millimetre and a half above the free margin of the lid. The sutures are introduced a little to the conjunctival side of the lashes, brought out just at the lower edge of the wound made by the excision of the strip of skin, re-entered at the upper edge of the wound, passed deeply under the muscle, and made to emerge through the skin a centimetre or more above the wound. When the stitches are tied, the skin-wound is closed and the loosened lid-margin is tilted and everted by rotating it on its long axis. The effect is increased by turning the lashes upward against the skin of the lid and fixing them there by means of collodion. The stitches are removed the next day. Fig. 8 represents a vertical section showing the incisions of the cartilage and of the skin, and the placing of a suture. The thread after its second introduction should pass more deeply than represented in the illustration—beneath the muscle.

Fig. 8.



Grooving of the anterior surface of the cartilage is the basis of the operations of Streatfeild and Snellen. The following is Streatfeild's description of his operation: "An incision with a scalpel is made of the desired length, just through the skin along the palpebral margin, at a distance of a line or less, so as to expose but not divide the roots of the lashes; and then just beyond them the incision is continued down to the cartilage (the extremities of the wound are inclined towards the edge of the lid); a second incision, further from the palpebral margin, is made at once down to the cartilage, in a similar direction to the first and at a distance of a line or more, and joining it at both extremities; these two incisions are then continued deeply into the cartilage in an oblique direction towards each other. With a pair of forceps the strip to be incised is seized and detached with the scalpel."[1] The effect of this operation was to remove a wedge-shaped strip consisting of skin, muscle, and cartilage. The wound was left to cicatrize without stitching, and the contraction of the cicatrix drew the margin of the lid outward. The object of inclining the extremities of the wound towards the edge of the lid was to isolate the marginal portion of the orbicularis. Fig. 9 represents the lid held in a Desmarres clamp.

Fig. 9.



After Streatfeild.

[1] Royal London Ophthalmic Hospital Reports, vol. i. p. 125, 1858.

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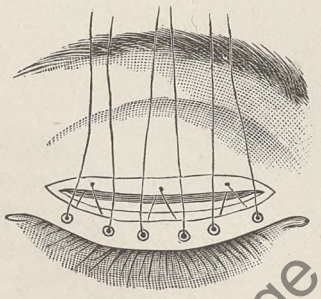
Snellen after forming a wedge-shaped groove in the cartilage brought its two surfaces in contact by sutures, without depending upon the effects of cicatricial contraction. The lid being held in a Snellen clamp, an incision was made through the skin only, parallel to the border of the lid and three millimetres above it. The upper edge of the wound was retracted, a band of orbicularis fibres about two millimetres in width was dissected out to expose the cartilage, and a wedge-shaped strip was cut from the latter, extending the whole length of the wound. This grooving was done by carefully made oblique cuts with a Beer cataract-knife, extending to the inner surface of the cartilage but not through it. Three stitches were then applied in the following manner. Each was armed with a needle at either end, and was entered into the cartilage above the groove and brought out at the upper margin of the latter; both needles were then carried to the lower margin of the wound, passed through the narrow band of skin containing the lashes, and made to emerge just above the edge of the lid, at a distance of about four millimetres from each other. A bead was passed on to each thread, and the suture was tightened by drawing upon it gently, while the beads were passed down by the closed blades of a pair of forceps, and tied. The ends of the thread were then placed against the brow and held there by means of plaster. The stitches

FIG. 10.



After Wecker.

FIG. 11.

After Wecker.

were removed on the third day. Figs. 10 and 11 are from Wecker and Landolt, and were made from drawings approved by Snellen.

Saunders,[1] believing that "such a vicious bending of the tarsus takes place that every attempt at re-establishing its original position must be fruitless," recommended its complete removal, and claimed to have obtained satisfactory results from the operation, which he described as follows: "A piece of thin horn, or a plate of silver, having a curvature corresponding to that of the eyelid, is to be introduced, and its concavity turned towards the globe, within the eyelid which is to be stretched upon it. An incision

[1] Treatise on Some Practical Points relating to Diseases of the Eye, 1816, p. 84.

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is to be made through the integuments and orbicularis palpebrarum, immediately behind the roots of the cilia, to the tarsus, and should extend from the punctum lachrymale to the external angle. The exterior surface of the cartilage is then to be dissected until the orbital margin is exposed, when the conjunctiva is to be cut through directly by the side of the tarsus, which must now be disengaged at each extremity, the only caution necessary being to leave the punctum lachrymale uninjured." He sometimes combined this operation with excision of the lid-margin. Such heroic measures may, fortunately, be considered out of date, and only in a case that had been very long neglected or very badly treated could the question of excising the entire cartilage arise. Still, even this is possible, and a familiarity with the different expedients that have been resorted to from time to time may be useful in suggesting modifications to meet special indications.

OPERATIONS FOR ECTROPION.

Acute ectropion resulting from inflammatory swelling of the conjunctiva is usually corrected spontaneously as the conjunctivitis subsides. It is recommended to keep the lids in position by means of compress and bandage, but this will often be found impracticable on account of the free discharges. Scarification of the chemosed conjunctiva is useful; it is best accomplished by introducing one blade of a pair of sharp-pointed scissors beneath the membrane and slitting it freely throughout its whole extent. This operation may be several times repeated, if necessary. The orbicular spasm may be relieved by canthotomy. If the lids are kept permanently everted by hypertrophy of the conjunctiva, a portion of the latter should be excised. This may be done with the sharp-pointed scissors, or the lid may be held in a Snellen's clamp and a fold of the thickened conjunctiva be dissected out with a knife. In simple removal of the margin of the lower lid from contact with the ball, due to senile relaxation or partial paralysis of the orbicularis, in which the chief inconvenience results from eversion of the punctum and consequent epiphora, much relief may be obtained by slitting the canaliculus. To prevent reunion of the incision and establish a permanent exit for the tears, the posterior lip of the wound should be seized with the conjunctival forceps and the margin be cut away with a small pair of scissors. This converts the canaliculus into a groove, extending well back into the canthus, which takes up the tears. In these cases the appearance is often much improved by narrowing the commissure. The edges of the lids at the outer canthus are freshened and brought together by a suture (tarsorraphy).

In ectropion of the lower lid without cicatricial contraction and without much elongation of the lid-margin, Snellen's operation by ligature will often give good results. Each end of a thread is attached to a needle, and both needles are entered through the conjunctival fold and brought out through the skin two centimetres below the margin of the lid. The points of entrance should be a half-centimetre and the points of exit a centi-

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metre apart. Traction is made upon the ends of the thread until the lid is replaced, and they are then tied over a roll of chamois-skin. Two threads may be used, if necessary. A compress bandage is applied, and the threads are allowed to remain for four days. The same effect is produced more certainly and permanently by an operation proposed by Dieffenbach.[1] An incision is made through the skin parallel to the lower margin of the orbit and a few lines above it. The wound is made to gape by stretching, and the dissection is continued until the conjunctival fold is reached. The conjunctiva is then incised throughout the extent of the wound, and the anterior edge of this conjunctival incision, which is connected with the attached margin of the tarsus, is drawn into the wound by hook or forceps and secured there by sutures.

When there is considerable elongation of the lid-margin it is necessary to remove a portion of it, and a number of operations have been practised for this purpose. One of the simplest is that known as Sir William Adams's. A wedge-shaped piece, involving the whole thickness of the lid, is excised at the middle of the lid-margin and the edges of the wound are brought together by small harelip pins. (See Figs. 12 and 13.) A simi-

FIG. 12.

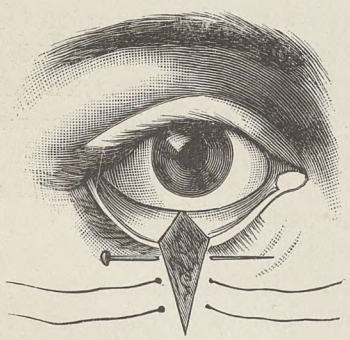
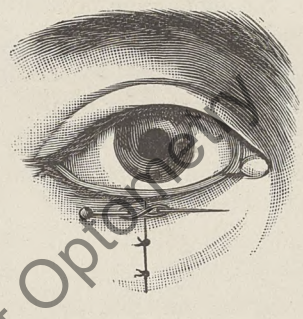


FIG. 13.



lar operation was performed fifteen centuries ago by Antillus, who removed an A-shaped segment including the conjunctiva, cartilage, and muscle, but leaving the skin intact.[2] The healing of a wound in this situation is in danger of being interfered with by the accumulation of tears, and a puckered or irregular perpendicular cicatrix in the middle of the lid is a serious deformity. The union is more prompt and sure and the final effect much better if the redundant lid is shortened by removing a piece at the outer canthus, as was suggested by Von Ammon.[3] Figs. 14 and 15 illustrate this method of operating.

The following operation has been devised by Dieffenbach. A triangular

[1] Wecker et Landolt, t. i. p. 198.
[2] Mackenzie, Am. ed., p. 237.
[3] Zeitschrift für Augenheilkunde, Bd. i. S. 529.

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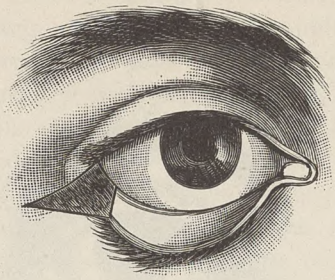
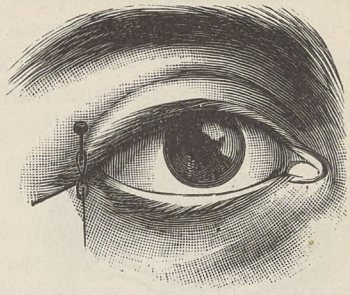
flap of skin is first excised at the outer canthus. Its base, six or eight millimetres long, is continuous with the commissure, and its apex points

Fig. 14.



After Von Ammon.

Fig. 15.



After Von Ammon.

downward, $AA'B$. (Fig. 16.) The canthus is then divided, and an incision $A'A''$ is made on the conjunctival surface of the lid-margin, equal in length to the base of the triangle. The triangular flap thus marked out is dissected up and slit outward, so that, as shown in Fig. 16, A' is carried to A and A'' to A', and is held in its new position by four sutures. Fig.

Fig. 16.

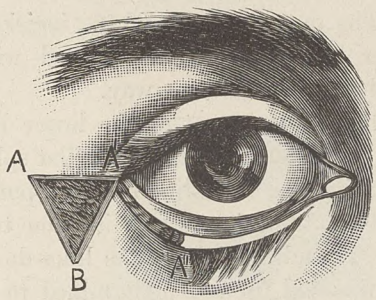
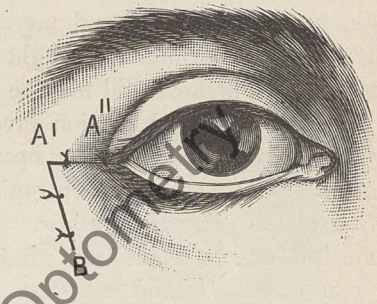


Fig. 17.



17 shows the new positions occupied by A'' and A'. In case of failure of union by first intention, the cicatricial contraction of the granulating surface tends to correct the ectropion by dragging the margin of the lid outward.

When the eversion is due to contraction of the skin, as so frequently is the case after burns, some proceeding must be adopted that will return the integumental surface of the lid to its normal position at the expense of the skin of the neighboring regions. If the skin of the whole lid has been destroyed, its restoration is undertaken by means of some of the more extensive operations described under Blepharoplasty. (See page 109.) For cases in which a portion of the lid is involved, or in which it is dragged out of position by cicatrices situated beyond it, a great number of operations have been proposed. Only a comparatively small number of these will be described to illustrate the principles of treatment, as it will be found in practice that each case is, to a great extent, a law unto itself, requiring special adaptations that tax the ingenuity of the surgeon.

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One of the most frequently performed is the transposition of a triangular flap, as suggested by Wharton Jones. Figs. 18 and 19 illustrate the

FIG. 18.

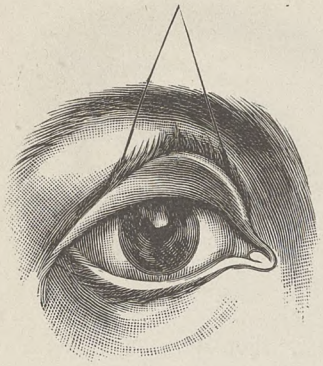
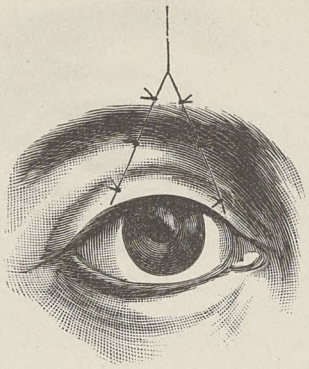


FIG. 19.



operation as performed on the upper lid, for which it was first proposed. V-shaped incisions are made including the skin of the everted portion of the lid, the margin of which forms the base of the triangle. The flap is then pushed downward until the lid is replaced by simply stretching the subcutaneous tissue, if this is possible, or by dissecting up the skin to the extent that may be found necessary. The outer margins of the incisions are then undermined, brought together by stretching, and united with sutures. The lower lid may be operated upon in the same way.

For extensive ectropion involving the whole extent of the lower lid, Graefe devised the following operation. The margin of the lid is first split, as in Arlt's operation for entropion, by an incision extending from the punctum to the external canthus. From the extremities of this incision two vertical cuts are made through the skin, extending eight or ten lines down upon the cheek, and a square flap is dissected up and undermined to its base. This flap is then forcibly stretched upward and held in place by

FIG. 20.

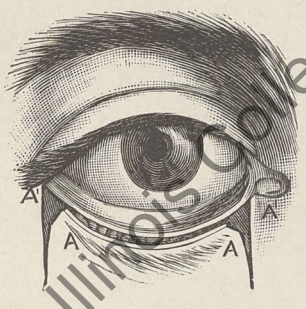
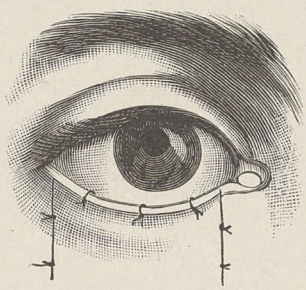


FIG. 21.



sutures, commencing at the lower extremities of the vertical incisions. To shorten the elongated lid-margin and more effectually raise the flap, the angles of the latter are bevelled by an obtuse-angled incision, *A*. When

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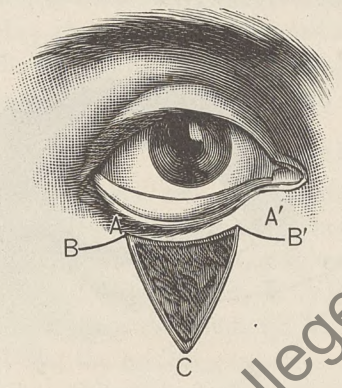
the edges of the wound are brought together, A is carried to A'. (Figs. 20 and 21.)

The operation of Jaeger[1] has for its object the replacing of the skin of the eyelid by means of extensive undermining and stretching of the neighboring integument. It is applicable to cases of considerable loss of substance in the skin of the lid, either with or without an adherent cicatrix. In case of cicatricial contraction of the upper lid drawing its ciliary margin to the upper edge of the orbit, he proceeded as follows. The lid was first freed by a deep incision made parallel to its margin and about midway between it and the superciliary ridge. The bridge of tissue thus formed, including the lid-margin, was reduced to its proper length by excising a quadrilateral piece from its centre and suturing the wound. Then, any adhesions that existed having first been freed, the upper edge of the incision was drawn forcibly forward, a long straight bistoury was inserted between the orbicularis muscle and the frontal bone, and the integuments were separated over an area extending from the temple to the middle line of the forehead. The skin and muscle covering the supraorbital region and the angles of the orbit, loosened in this way, were slid over the eyeball to form a new lid, and the edges of the horizontal wound were brought together by sutures.

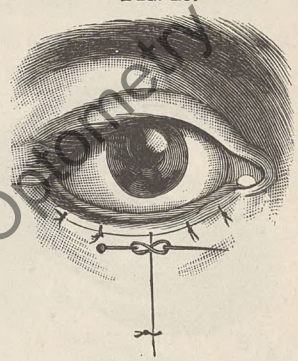
If the cicatrix that causes the ectropion is very dense and firmly adherent to the bone, it may be necessary to excise it and fill the space that it occu-

FIG. 22.



After Zeis.

FIG. 23.



After Zeis.

pied by neighboring integument, as in the following operation by Dieffenbach.[2] The cicatrix is included in a triangular incision, the base of which is a little below the everted lid-margin and parallel to it, AA' (Fig. 22), and is dissected away. The base-line is then extended by two other incisions, AB and $A'B'$, having the same curve as the first, and the flaps CAB and

[1] Brown, London Medical Gazette, vol. xvii. p. 721.
[2] Zeis, Handbuch der plastischen Chirurgie, 378.

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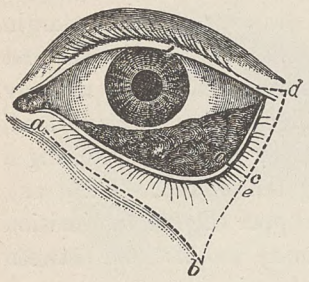
$CA'B'$ are undermined and their edges united so that A and A' are brought together in the middle of the base-line. (Fig. 23.)

A small cicatrix may be left in place and covered over by the neighboring integument, instead of being excised.[1] It is circumscribed by two elliptical incisions, and its surface is freshened. The integument on each side is then undermined sufficiently to allow the lid to be brought into proper position, and is united in front of the scar, which is thus buried.

Fig. 24.



In cases of ectropion due to adherent cicatrices resulting from caries of the orbital margin, Arlt[2] operated as shown in Fig. 24, in which the cicatrix is in the neighborhood of e. The incisions ab and bc are made through the skin and muscle; the lid-margin, with cilia bulbs, is excised from e to d; the triangular flap abc is dissected free by incisions carried back to the posterior edge of the tarsus; and the lid is replaced by bringing c to d. The space left bare by sliding up the flap is reduced, so far as possible, by stretching the skin horizontally and suturing the margins.

Richet[3] has described a very ingenious operation for ectropion of the lower lid with dragging downward of the external canthus due to an adherent cicatrix at the outer margin of the orbit. The cicatrix is included in the incisions ADC and $AB'C$ (Fig. 25) and dissected out with the

Fig. 25.

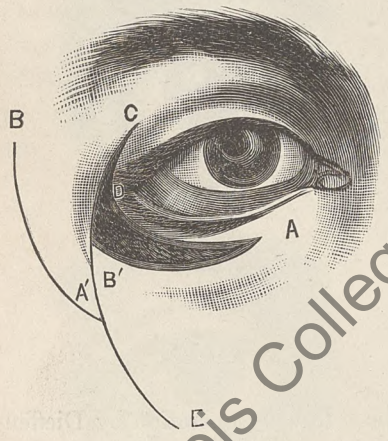
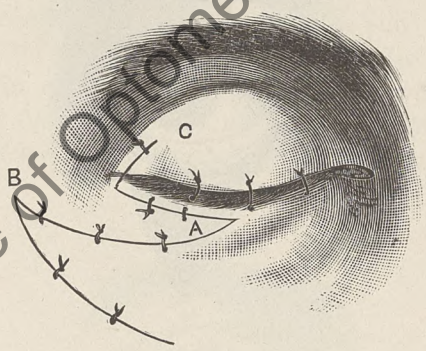


Fig. 26.



skin thus outlined. The incision CB' is continued to E, and another flap is marked out by an incision $A'B$. This flap, $CA'B$, is then freed and transplanted to the space bared by the excision of the skin containing the

[1] Von Ammon, Zeitschrift für Ophthalmologie, Bd. i. S. 49, 1831.
[2] Graefe und Saemisch, Bd. iii. S. 463.
[3] Recueil d'Ophtalmologie, 1873.

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cicatrix, so that A' is carried to A. (Fig. 26.) The flap $AB'E$ is also dissected free, and is drawn into the space from which the other flap was taken, so that B' is carried to B. The margins of the upper and lower lids are united by a temporary ankyloblepharon.

In ectropion following blepharitis, Fukala[1] operates as follows. The skin and muscle are dissected from the cartilage throughout the whole extent of the lid (lower). A thread is introduced through the skin three or four millimetres from the external canthus and four or five millimetres below the free margin of the lid into the space separating the skin and muscle from the cartilage, passed through the cartilage at a point near its upper border, reintroduced through the cartilage three millimetres to the nasal side of the first point, and brought out through the skin by the side of its place of entrance. The two ends of the thread are then tied over a roll of cotton and allowed to remain in place for four days. A similar suture is placed near the internal canthus. The object of the operation is to depress the cartilage, and at the same time raise the skin and muscle. To remedy the lengthening of the lid-margin a flap of skin is taken from the external angle. In senile and conjunctival ectropion he splits the lid-margin and excises all the ectropinized parts, conjunctiva and cartilage.

To meet the indications presented by special cases, it will often be necessary to combine several features of the different operations described. When, for instance, a decidedly elongated lid-margin is replaced it may require to be shortened by tarsorraphy or excision. In case of the absence of available sound integument from which to form a flap, or of its loss by sloughing after operation, recourse may be had to the introduction of dermic or epidermic grafts, or to the transplantation of flaps from distant parts, as the arm. (See Blepharoplasty.)

BLEPHAROPLASTY.

Blepharoplasty may be considered a comparatively recent operation. Until the present century the aphorism of Celsus, "Si palpebra tota deest, nulla id curatio restituere potest," was universally accepted, and in 1814 Boyer, in his classical work on surgery, strongly advised against operative interference when the whole lid was involved in malignant disease, on the ground that the resulting exposure of the eye was worse than the original condition. Others recommended the removal of the eyeball to save the patient the suffering that would result from its exposure.[2]

The credit of first forming a new lid is due to C. F. Graefe, who, however, merely refers to the operation incidentally and gives no details. Writing in 1818,[3] he states that several years before he had performed an operation for the restoration of a lower lid destroyed by ulceration, transplanting a flap of skin from the immediate neighborhood. He adds that,

[1] Annales d'Oculistique, January, 1894, p. 43.
[2] Edinburgh Medical and Surgical Journal, 1832, p. 254.
[3] Rhinoplastik, S. 15.

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being without any antecedent, he performed the operation under grave apprehensions, but that it was followed by complete success.

About the same time Dzondi[1] reports a case in which he restored the greater part of a lower lid by a flap taken from the malar region. It is difficult to understand from the long and complicated description without illustration exactly what was done, but the flap seems to have been formed of the skin immediately below the bared space, retaining an attachment at the inner canthus, and to have been slid upward into place. There is some doubt about the success of the procedure, as a number of subsequent operations were done upon the patient. Dzondi speaks of having performed several similar operations before.

The subject was allowed to rest for more than a decade, when it was revived by the operations described by Fricke, Jungken, and Hysern y Molleras in 1829. Then came Dieffenbach's brilliant operation in 1835, which some authors claim was the first complete restoration of the whole lid and which seems to have established blepharoplasty on a firm basis and given it a universally recognized position in surgery. Since then there has been no reason to complain of the number of inventors and modifiers who have engaged in the work, though Wecker intimates that the scalpel has been used less than the pencil and pen.

The methods that have been resorted to for forming a new lid, or restoring one partially destroyed, may be classified as follows:

I. Transplantation of a flap from the neighboring skin with a pedicle or a broad base, with more or less twisting of the latter.

II. Direct displacement of a flap from the adjacent skin by a sliding movement.

III. Transplantation from a distant region of a flap with a pedicle.

IV. Transplantation of a number of small epidermic or dermic "grafts."

V. Transplantation from a distant region of a flap of skin without pedicle, or of an epidermic flap.

I. The first method was borrowed from rhinoplasty, and is known in plastic surgery as the Indian method. The early operations of Graefe and Dzondi belong to this class. Jungken, in attempting to form a lower lid by a flap taken from the cheek,[2] followed the Indian plan exactly and left a bridge of sound skin between the incision for the flap and the bared space, over which the pedicle was passed. After the new lid had united the pedicle was to be cut and turned back to cover the space from which it had been taken. Two operations performed in this way were acknowledged failures. Fricke soon afterwards was more successful. He modified the operation by giving the flap a broad base and connecting it continuously and permanently with the bared space. "When the flap is in

[1] Hufeland's Journal, 1818, p. 100.

[2] Arch. Gén. de Méd., 1829, t. xxvii. p. 257.

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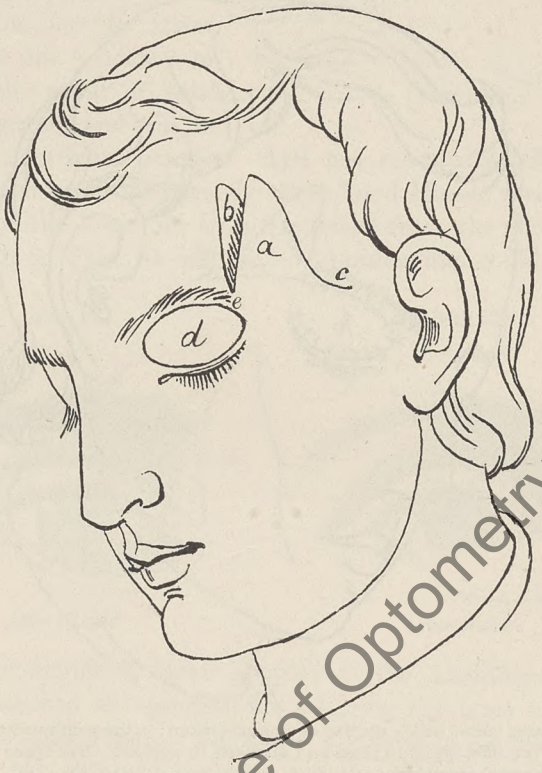
place there still remains a bridle of skin between the internal incision of the flap and the external canthus. A piece of this is removed large enough to allow the flap to be accurately fitted." He took his flap for the upper lid from the temple and forehead, "a little external to and two lines above the margin of the orbit," and from the malar region for the lower lid. The flap was made one line larger in all directions than the space to be covered. As this operation is generally incorrectly described, exact copies of Fricke's original plates are given (Figs. 27, 28), with the following explanation in his own words:[1]

FIG. 27.



"*a*, the flap of skin dissected up; *b*, the space from which the flap was taken; *c*, the incision carried further outward to facilitate the turning of the flap; *d*, the upper eyelid separated by an incision and drawn apart; *e*, the interspace which, before the placing of the new eyelid, must be cut through, and out of which a piece of skin of sufficient size must be taken; . . .

The patients upon whom he operated were cases of extreme ectropion in which the skin only of the lid was destroyed, the lid-margin and the conjunctiva remaining sound.

Hysern y Molleras, of Madrid, in a publication entitled "Tratado de la blefaroplastia temporofacial, ó del método de restaurar las destrucciones de las párpadas," which appeared in 1834, described an operation for restoring the upper lid performed in 1829. The original is not accessible,

[1] Die Bildung neuer Augenlieder, 1829.

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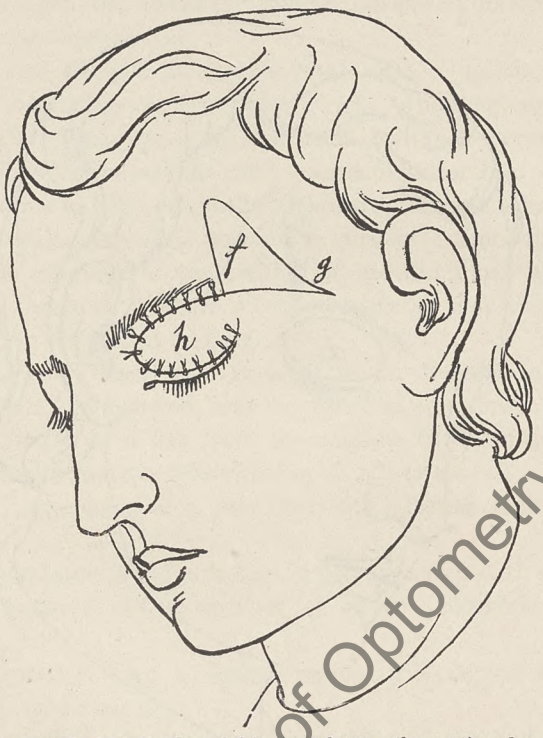
but from reference to it by Serre the plan of the operation seems to have been much the same as that followed by Fricke. Hysern y Molleras made a point of including muscle in the flap. The operations described by Fricke, who published the first treatise on the subject, may be considered the basis of all procedures for blepharoplasty by the Indian method. Many modifications have been introduced to meet the requirements of different cases and the views of different surgeons, and there must necessarily always be more or less individuality in every operation, as the conditions con-

FIG. 28.



". . . *f*, the space out of which the flap of skin was taken; *g*, the continued upper wound-margin of the new lid; *h*, the new eyelid in place and attached by sutures." The space denuded by removal of the flap was allowed to granulate.

stantly vary. All surgeons now place the base of the flap immediately adjacent to the bared space, or, if necessary, prolong the latter to the base of the flap, which is practically what Fricke did in cutting away the piece of sound skin left between them. According to Billroth, Szymonowsky, and others, the wound made by dissecting away the flap should be closed before the flap is stitched in place, as the outlines of the space to be covered by the latter may be more or less altered by the stretching to which the neighboring skin is subjected.

Blasius restored a lower lid by a flap taken from the side of the nose. This flap was included between two crescentic incisions to give a form to the secondary wound that admits of its edges being brought together more

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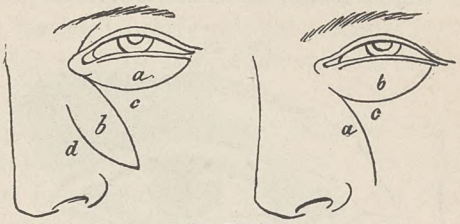
readily. Blasius has been incorrectly quoted by several authors (Meyer, Wecker) as taking his flap for the lower lid from the glabella and forehead. Figs. 29 and 30 are reproduced exactly from the original article,[1] in which no mention is made of any other operation except a suggestion that the upper lid might be restored by a similar flap taken from the root of the nose and the glabella.

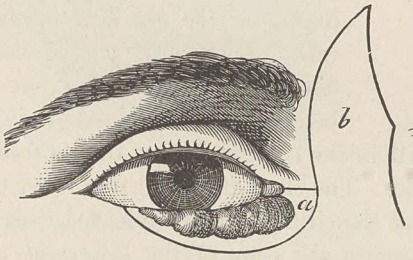
Fig. 29. Fig. 30.



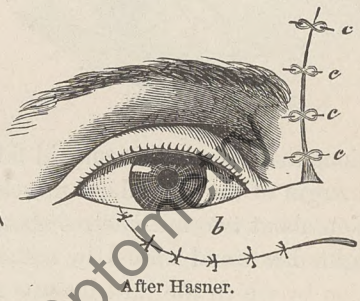
After Blasius.

Velpeau preferred to take the flap from the malar region for the upper lid, and from the temporal region for the lower lid, to obviate the tendency to ectropion as the result of subsequent contraction, and this is Dr. Knapp's invariable practice. Arlt has repeatedly taken a flap for restoration of the upper lid from the cheek,[2] and Hasner restored the inner three-fourths of the lower lid by a flap taken from the glabella and forehead, as shown in Figs. 31 and 32.[3] A similar flap could, of course, be

Fig. 31. Fig. 32.



After Hasner.



After Hasner.

taken from the temple if better adapted to the conditions of the case. Hasner also described an operation for restoring the inner angles of both lids in a case of epithelioma. The parts involved in the disease were circumscribed by two elliptical incisions and dissected out, and to cover the resulting bared space a flap was formed from the skin of the side of the nose. The base of this flap was separated from the inner angle of the bared space by a bridge of sound skin three lines wide, and its free extremity was notched to form a canthus. The intervening bridge was now cut through and dissected up to form a second flap, which was slid downward and inward to partly fill the wound resulting from the removal of the first flap and to facilitate the bringing together of its edges. It does not seem clear why the fresh edges of the new canthus failed to unite, form-

[1] Medicinische Zeitung, März, 1842.

[2] Graefe u. Saemisch, Bd. iii. S. 471.

[3] Entwurf einer anatomischen Begründung der Augenheilkunde.

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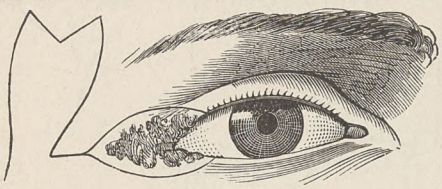
ing an anchyloblepharon; but the author states that he saw the patient two years after the operation and the angle was nearly perfect. The outer canthus may, of course, be restored in the same way by taking a flap from the temple or the cheek, as illustrated in Figs. 33 and 34.

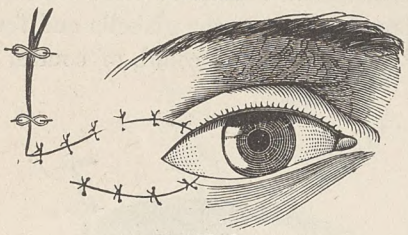
It too often happens that the region from which the flap is to be taken is not a matter of choice, but that the surgeon must "cut according to his cloth," and get sound skin wherever he can find it. In a case reported by Dr. St. John,[1] the patient's hair had been caught by a revolving shaft in a mill, and her entire scalp had been torn off, from forehead to occiput. The vast granulating surface was cicatrized by the aid of skin-grafting, but the contraction that resulted produced an extensive ectropion. There was no question here of utilizing the skin of the temple or forehead; but, instead of taking a flap from the cheek nearly vertically downward, as is usually done, Dr. St. John took one from below the lower lid, so placing the incisions that the cicatrix should fall in the natural furrow that is found at the margin of the orbicularis muscle. The edge of the lid was freed by an incision about two millimetres above the ciliary border, dissected loose, and brought down and united by sutures to the edge of the lower lid. The incision by which the lid-edge was freed was extended outwardly to a point about three centimetres from the external canthus, and from this point a curvilinear incision was carried, following the natural fold and keeping about two centimetres from the edge of the lower lid. This incision extended to the side of the nose, and then, turning sharply upon itself, ran in a slightly crescentic curve (concavity upward) below its former course, and distant from it, at a point exactly below the centre of the lid, two or three centimetres, corresponding to the width of the gap to be filled. This incision was prolonged in the direction of the ear at least one and one-half inches beyond the external canthus, and an incision from the upper edge of the gap left when the lid was dissected free was also carried outwardly parallel to the one just described. The tongue-like flap below the eye was then dissected up, and the dissection carried at least one inch beyond the canthus. The flap was then laid in the gap to be filled and secured by fine sutures. The large gaping wound upon the cheek was filled by undermining with

Fig. 33.



After Wecker.

Fig. 34.



After Wecker.

[1] Trans. Am. Ophth. Soc., 1893, p. 597.

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scissors the lower edge only, and this undermining had to be very extensive, —at least two inches,—in order to have as little tension as possible. (Figs. 35 and 36.)

The lids were left united for several weeks. The result was very satisfactory, the scar of the secondary wound being scarcely visible.

I have restored the upper lid by a flap similar in form to the one just described, but taken from the forehead nearly parallel to and a little above the eyebrow. The cicatrix fell into the horizontal wrinkles of the forehead and could hardly be noticed.

Some years ago I assisted the late Dr. Joseph Pancoast in an operation upon a patient suffering from the result of an extensive burn. The skin of the upper lid was destroyed, and its margin was adherent to the edge of the orbit; there was an extensive cicatrix of the forehead, and the eyebrow had entirely disappeared. He transplanted a flap taken from the

Fig. 35.

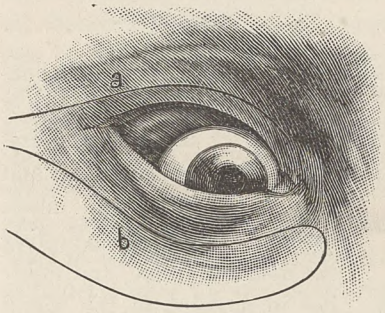
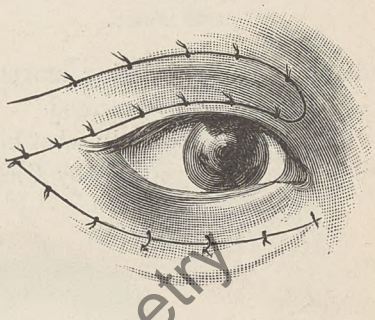


Fig. 36.



skin far back in the temporal region, including a narrow strip of the scalp in its outer edge. When the flap was in place the hair on its upper margin was made to take the position of the eyebrow.

Landolt[1] describes an operation for replacing the lower lid by a flap taken from the upper. The whole of the lower lid, except a part of the conjunctiva, was removed in the extirpation of a carcinoma. What remained of the conjunctiva was adherent to the orbital margin, and the skin below it was cicatricial from a previous unsuccessful operation. The conjunctiva was freed from the orbital margin and dissected up back to the ball. An incision was made in the upper lid parallel to its margin and two millimetres above it, extending at each end beyond the canthus and carried down to the cartilage; and a similar incision parallel to the first and seven millimetres above it. (Figs. 37 and 38.) The flap thus marked out, and including the fibres of the orbicularis muscle, was freed, except at the ends, where broad attachments were left, and was brought down to take the place of the lower lid. The upper border was stitched to the conjunctiva, and the lower to the margin of the skin. (Fig. 38.) The edges

[1] Arch. d'Ophtalmol., 1885, p. 492.

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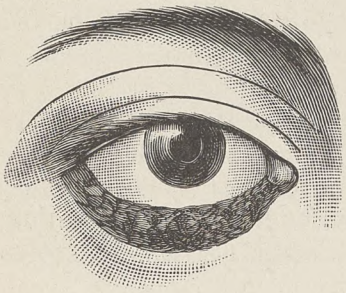
of the secondary wound in the upper lid were brought together and sutured. Some weeks later, when union was complete, the pedicles were cut and placed in better position at the canthi.

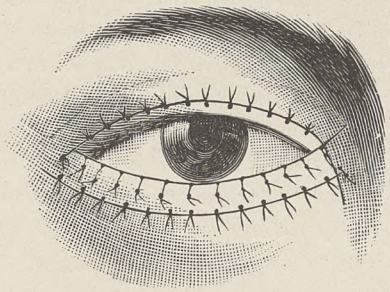
The second method, or that by direct displacement of a flap from the

FIG. 37.



After Landolt.

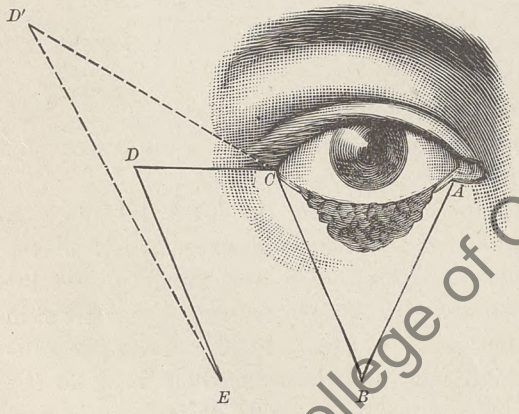
FIG. 38.



After Landolt.

adjacent skin by a sliding movement, is sometimes known in plastic surgery as the "French method." A hint of it is given by Celsus, though it is not quite clear exactly what his procedure was.[1] This method was first applied to blepharoplasty by Dieffenbach in 1835.[2] Two incisions, *AB* and *BC* (Fig. 39), were made through the skin, including the diseased tissue, and meeting in a point in the sound skin below it, and the triangle thus marked out was excised. An incision *CD* was then made horizontally outward from the canthus, and from its extremity another incision *DE* downward and parallel to *BC*. *CD* should be rather longer than the base of the triangle. The flap *CDEB* was dissected free to its base *BE*, and transferred by a sliding movement that brought *C* to *A* and *D* to *C* (Fig. 41), and was retained in its new position by sutures. The triangular space *CDE*, which was now denuded of skin, was left to granulate.

FIG. 39.



This operation has undergone a number of modifications from time to time. The incision *DE* is usually carried lower down on the cheek than

[1] Greive's translation, p. 411.
[2] Casper's Wochenschrift, Bd. i. S. 8.

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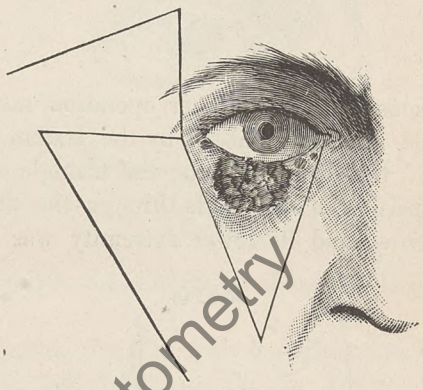
CB, to enable the flap to be transferred with less torsion of its base. Arlt makes this incision *DE* convergent instead of parallel to *CB*, so that the base *BE* of the flap is only about half as long as the top *CD*.[1] Szymonowski,[2] instead of making the incision *CD* horizontal, carries it upward and outward to *D'*, so that the flap ends in an acute angle, as represented by the dotted lines. This gives a fuller flap and leaves a secondary wound whose edges can be more closely approximated. It also lessens the tendency to drawing down of the canthus by subsequent contraction.

The displacement of the canthus and the stretching of the new lid by the cicatrization of the large space left to granulate, in its usual position, are the most serious drawbacks to the Dieffenbach operation. This contraction may be to a certain extent diminished by skin-grafting, or by the transplanting of a Wolfe or a Thiersch flap, but it is well known that it cannot in this way be entirely prevented. In a case in which an epithelioma involved the whole of the lower lid and measured an inch in its horizontal and three-fourths of an inch in its vertical diameter, I modified the operation by filling the space from which the flap was taken by another flap formed from the skin of the temple, thus leaving the granulating surface above the line of the canthus. The bared triangle was first diminished in size as much as practicable by undermining the skin and bringing its edges together at the lower and outer angles; and the same thing was done with the second triangle left on the temple. Fig. 40 shows the lines of the incision, Fig. 41 the position of uncovered space in Dieffenbach's operation, and Fig. 42 its position in the modified operation. Another advantage gained is that the nutrition of the new lid is better maintained when both of its edges are stitched to sound skin than when one of them is left to form the margin of an extensive granulating surface. Three years afterwards the canthus was in normal position and the deformity was very slight.[3]

FIG. 40.



Dieffenbach's method may be applied to the restoration of the upper lid by taking the sliding flap from the forehead and temple. In many cases some other plan will be preferred to avoid disturbing the eyebrow. Where only a part of the lid is destroyed, the flap may be formed from the skin of the lid only, below the eyebrow, as has been done by Landolt.[4]

[1] Graefe u. Saemisch, Bd. iii. S. 475.
[2] Handb. der Oper. Chir., p. 223.
[3] Trans. Amer. Ophth. Soc., 1891.
[4] Archives d'Ophtalmologie, 1885.

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In a case of epitheliomatous disease involving the inner third of the upper lid and the inner angle of the lower, and extending to the side of the nose-bridge and quite deeply back into the orbit, I operated as is shown in Fig. 43. An extensive cicatrix of the side of the nose, the result of cauteriza-

Fig. 41.

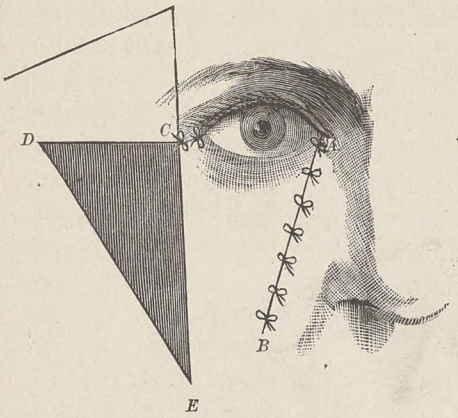
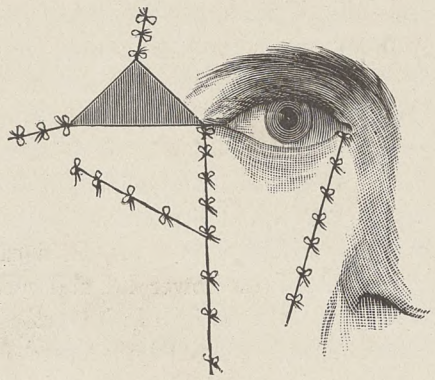


Fig. 42.



tions and of a former operation, made it impossible to take a flap from the cheek or glabella by the Indian method. After complete extirpation of the diseased parts, the triangle of skin *abc* was also removed. The incision *ed* was made through the skin of the temple, external to the eye-brow, and its lower extremity was connected with *a* by an incision *da*

Fig. 43.

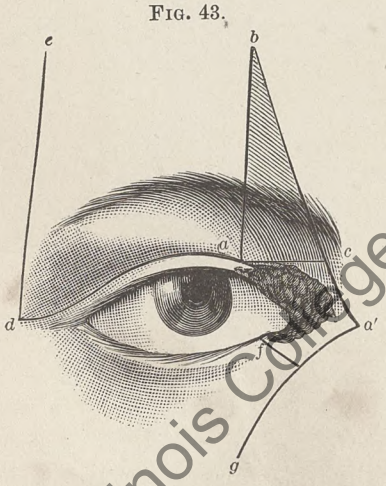
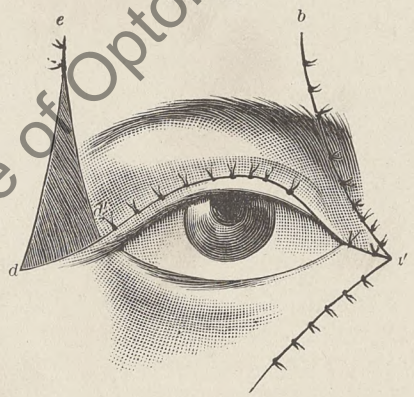


Fig. 44.



carried through the skin only of the lid just above the lid-margin. The flap *bade* was dissected free to its base, and, by a sliding movement, *a* was brought to *a'* and *d* to *d'*. (Fig. 44.) The lower lid was then freed by an incision *fg* downward and outward upon the cheek, and was drawn up to be stitched to the upper flap at the canthus.

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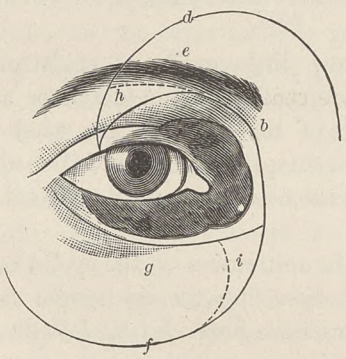
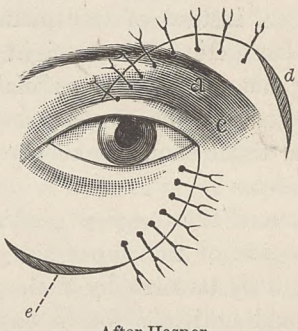
Figs. 45 and 46 show the lines of incision made by Hasner[1] for the formation of sliding flaps with curved margins. The points of the flaps were cut off, as shown by the dotted lines at *h* and *i*, and the free end of the lower was united with the base of the upper. The patient was the subject of an extensive epithelioma involving both lids and extending beyond the canthus and into the orbit. The nasal side of the eye was denuded of conjunctiva in extirpating the growth, and the new lids became adherent to the ball, but sight was preserved. If the lids had not united to the ball, it would have been difficult, if not impossible, to prevent them from uniting with each other, as the margins of both were fresh. This operation affords a good illustration of what may be done by curved sliding

FIG. 45.



After Hasner.

FIG. 46.



After Hasner.

flaps. Either of them could be adapted to the restoration of one lid if the other lid were sound.

Transplantation from a distant region of a flap with a pedicle, known as the Italian or Taliacotian method, had its origin, like the Indian, in mutilations of the nose as a punishment. Attempts at restitution of the lost member were never forbidden, as it was held that the victim suffered additional punishment at the hands of the surgeon and took serious chances of failure. Tagliacozzi was not the inventor of this method, which was practised before him by the Sicilian Brancas, but he wrote the first systematic work on the subject, in 1597. The flap is taken from the arm or hand, and, until union has taken place, the arm is bound to the head by bandages or straps. The operation is tedious and painful, the suffering caused by the constrained position maintained for many days being serious, and it would now scarcely be considered unless in exceptional cases. It has not been frequently applied to blepharoplasty and is not usually mentioned in ophthalmic text-books, but has sometimes been found available as a last resource in cases of extensive destruction of the skin of the face by burns. Such a case is reported by Dr. R. H. Derby.[2]

[1] Loc. cit.

[2] Trans. Amer. Ophth. Soc., 1885, p. 141.

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The method of transplanting small flaps of epidermis, or "grafting," was originated by Reverdin, in 1869.[1] He usually took his grafts from the inner surface of the leg. Pinching up a piece of skin on the inner surface of the tibia between his forefinger and thumb, he introduced the point of a lancet parallel to the bone and at a depth of about a millimetre, brought it out three or four millimetres beyond, and passed it on until the edges cut out a little flap of epidermis. The wound left was stippled with points of blood. He then placed the lancet, carrying the graft, on the granulating surface and with the point of a needle slid the graft in place, moving it a little back and forth to make sure that its edges were not turned in and that its deep surface lay evenly in contact with the granulations. When the desired number of grafts had been transplanted, he retained them with strips of diachylon plaster, which were not removed for twenty-four hours.

Ollier,[2] instead of transplanting these little grafts of epidermis, used larger flaps, from four to eight or more centimetres in diameter and including not only the superficial layers of the skin but the whole derm. He insists that the cicatrix should not simply be freshened, but that the inodular tissue should be removed, and the new flap applied to sound underlying tissue.

Lawson[3] first applied grafting to the restoration of the eyelid in a case of ectropion of the upper lid. After dissecting the lid-margin free and uniting it by tarsorraphy to the margin of the lower lid, he left the resulting wound until the fourth day, when it was covered with healthy granulations, and then transplanted a piece of skin "the size of a threepenny piece, and two days later another portion the size of a silver fourpenny. Both pieces rapidly united to the granulating surface, and the space between them was speedily filled up with new cicatricial tissue."

Wecker[4] covered the whole granulating surface completely with a "mosaic" of small dermic grafts taken from the forearm or arm. He pinched up a little fold of skin with the finger and thumb, transfixed it with a small bistoury, and freed it with a pair of curved scissors, forming a flap which after contraction measured six or eight millimetres in diameter. When a sufficient number of such grafts had been transplanted they were covered with gold-beater's skin, and over this a retaining compress and bandage were placed. In cases of ectropion, after freeing the lid-margin and uniting it with the other lid by tarsorraphy, the gaping wound was left for seven days to form a granulating surface before the grafts were transplanted. The lid-margins were allowed to remain united for several months. Later,[5] he advised the immediate transplantation of the grafts

[1] De la Greffe épidermique.
[2] Bull. de l'Acad. de Méd., 1872, p. 243.
[3] Lancet, November 19, 1870.
[4] De la Greffe dermique en Chirurgie oculaire, Annales d'Oculistique, 1872.
[5] Wecker and Landolt.

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upon the fresh wound; if they do not live they can still be replaced by others after granulations have formed.

Transplantation from a distant region of a flap without a pedicle has been quite frequently resorted to. Bünger, of Marburg, in 1823,[1] restored a nose destroyed by lupus with a flap of skin taken from the thigh, with partial success. There is a legend that the Indians employed this method in their rhinoplastic operations several centuries ago,—that they transplanted flaps from the skin of the gluteal region, first increasing the circulation by palpation and friction; but the history of these operations is generally considered rather apocryphal. Butler had probably heard of them, but attributes them, in Hudibras, to Taliacotius, who from "the brawny part of porter's bum cut supplemental noses," etc. Le Fort, however, who made the first attempt to apply this method to blepharoplasty,[2] states that he was led to try the experiment by accounts that he had seen in the *Indian Annals of Medicine* of operations performed by the natives for the restoration of the nose by flaps taken from the skin of the buttock either of the patient or of any one else who might be willing to supply it for a consideration. He took a flap from the arm large enough to cover the whole of the denuded surface and retained it in place by seven or eight sutures and a light compress. It sloughed completely in a few days; a result which was attributed, perhaps correctly, to the fact that the transplanted skin was too thick. Two years later [3] he operated on another patient, taking care to free the flap completely of fat and subcutaneous tissue, and met with complete success. Successful cases were also reported by Sichel[4] and by Stellwag,[5] in 1874, but the method was not extensively adopted until brought to the attention of British surgeons by Wolfe.[6]

Wolfe reported two successful cases, and the operation has since been usually known by his name. They were cases of cicatricial ectropion in which the skin of the lower lids was destroyed but the conjunctiva and lid-margin remained sound. The lid was freed by an incision parallel to the margin and two lines below it, drawn up into position, and united to the upper lid. The space thus left bare was two inches long and one inch broad. The hardened cicatricial subcutaneous tissue was cleared away, and the margins of the wound were undermined so as to serve as a frame into which the new flap was to be inserted. The required flap was then removed from the forearm, carefully freed from fat and subcutaneous tissue, and applied to the gap, being so set that the old cicatricial skin overlapped its edges and answered the purpose of stitches. The whole was covered with fine gutta-percha tissue and over this a lint compress and bandage.

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[1] Graefe und Walther, Bd. iv. S. 569.
[2] Gaz. Hebdom., Mars, 1872, p. 140.
[3] Bull. de l'Acad. de Méd., 1872, 2e Série, t. i. p. 295.
[4] Ibid., 1875, t. iv. p. 574.
[5] Allgem. Wien. Med. Zeitung, 1874, n. 32.
[6] Med. Times and Gazette, 1876, vol. vi. p. 608.

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The dressing was not disturbed until the third day. Before this time but four or five cases had been reported, but the operation has been performed very frequently since in England, on the Continent, and particularly in America; so that Wolfe has the merit of bringing this method of blepharoplasty prominently before the profession, but whatever credit attaches to originating it unquestionably belongs to Le Fort. Wadsworth first performed the operation in this country in 1876,[1] and a number of other American surgeons have reported operations, a large proportion of which are recorded in the third volume of the "Transactions of the American Ophthalmological Society."

Comparison of the experiences and views of various operators suggests the following points as to the details of the operation:

The strictest antiseptic precautions should be observed before, during, and after the operation. The skin about the eye and in the region from which the flap is to be taken should be washed with soap and water and bathed with bichloride solution. To determine accurately the size of flap required, to secure the cessation of bleeding, and to avoid delay in transplanting the flap, the space to be filled is to be prepared first. In case of cicatricial ectropion, to which this operation has been usually applied, the lid is dissected free, placed in position, and securely united to the other lid. The surface of the gaping wound thus made should be freed, so far as possible, from cicatricial tissue until comparatively sound underlying tissue is reached. If bleeding does not cease spontaneously, it may be checked by hot water, and, if necessary, by pinching open vessels with the forceps or holding them in small clamps. The denuded space is then cleansed of clots and bathed with bichloride wash or sterilized salt water, and covered with gauze wet with the solution while the flap is being prepared. Some surgeons object to the use of the usual antiseptics upon surfaces to which flaps or grafts are to be applied, because of their coagulating effect. Thiersch recommends common salt, 6 to 1000.

The flap is usually taken from the inner surface of the arm or thigh, sometimes from the side of the chest. Jeffries[2] and Juler have suggested the prepuce as affording the best kind of skin for the purpose, as in fact it does, but there are practical difficulties, to say nothing of sentimental objections, in the way.

As there is very considerable immediate retraction of the skin when it is removed, the flap outlined should be about one-third larger than the space to be covered: some operators have made it nearly twice as large. I have found it a good way to take the size and form of a wound to lay a piece of thin tissue-paper upon it and cut out the part stained by its surface. This can be laid upon the skin and a line marked out with a scalpel or a pen parallel to its edges and at a proper distance from them. The flap can

[1] Trans. Fifth International Ophthal. Congress.

[2] Trans. Amer. Ophth. Soc., 1885, p. 113.

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then be cut out with a scalpel, or, as some operators prefer, with a pair of probe-pointed scissors. So far as possible the skin only is dissected out and is afterwards laid across the finger, while any remaining fat or subcutaneous tissue is carefully removed with a pair of scissors curved on the flat.

If the flap is found to be too large, it is slightly trimmed to enable it to lie in its new position smoothly without creasing or folding at its edges. Authorities vary as to the use of sutures, some avoiding them entirely and others using them freely. Generally the advice is to dispense with them so far as possible, and when the flap cannot be kept in place without them to use very fine antiseptic thread and insert it superficially. Ordinary sutures create points of suppuration. When the flap is carefully moulded in place the parts are covered with gold-beater's skin, or, as suggested by Fryer, with moistened gold-beater's-skin plaster. Over this iodoform may be dusted, or a few layers of iodoform or bichloride gauze may be placed, and the whole covered with a cotton compress and retaining bandage. The dressing should not be disturbed for four or five days, unless it is thought best to remove all but the transparent gold-beater's skin through which the condition of the flap may be noted.

Some surgeons merely hold the lid-margins together by sutures to secure immobility until the flap has united, while others unite them by tarsorraphy and do not separate them for several months or a year. In view of the strong tendency to contraction of the new lid, the latter plan would seem the more rational.

As to the final results of this method, it must be considered as still on trial. The important point to be decided is the extent to which the transplanted flap may be expected to contract. To determine this, reports of a considerable number of cases carefully observed several years subsequent to the operation are required; and these are wanting. Successful cases a few weeks after the operation, when they have generally been reported, leave scarcely anything to be desired; but the subsequent contraction in many has been excessive, and in some the flap has shrunk to a mere line or has apparently disappeared entirely by absorption. On the other hand, in a few cases that have been observed after comparatively long intervals the result has been very encouraging. In Sichel's case[1] the result remained excellent at the end of fifteen months. In Zehender's[2] the flap shrank for six weeks, but remained afterwards stationary for a year. In a case reported by Gruening[3] the flap contracted for a month, but nine months afterwards there was no further shrinkage. A case reported by Fryer,[4] however, shows that the last word cannot be said in reference to a flap transplanted from a distant part, even at the end of these long periods. A wound an inch and

[1] Loc. cit., 1875.
[2] Klin. Monatsbl. für Augenheilk., 1879, p. 213.
[3] Trans. Am. Ophth. Soc., 1883.
[4] Ibid., 1884–1885–1890.

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a quarter in length and three-quarters of an inch in width, left by the removal of an epithelioma of the lower lid, was covered by a flap of skin taken from the forearm. The flap as outlined was twice as large as the space to be covered, but fitted it accurately after the immediate shrinkage. Eighteen months later there had been "little or no retraction;" but when seen six years after the operation, the flap had contracted to one-third of an inch in its longest diameter and an ectropion had been produced.

In this connection the investigations of Garre[1] are interesting. Examining dermic grafts four or five months after transplantation, he found that the original vessels of the skin of the flap had undergone hyaline degeneration; while the new capillaries, proceeding from the neighboring parts and disposed in loops penetrating the derm, were surrounded with round cells and plasma to form granulations which were confounded with those of the raw surface. The new vascular loops advanced as far as the papillæ, but they were rare and did not form regular anastomoses as in sound skin. He concludes from these facts that the useful part of the graft is reduced to the deep layers of the epidermis, while the dermic layer is absorbed and is replaced by new connective tissue.

In the method known as Thiersch's,[2] the transplanted flap includes only the epidermis and a superficial layer of the derm. It is usually taken from the inner surface of the arm, which, as well as the wound to be covered, has been previously prepared as directed in the case of the skin-flap. While the skin is well stretched by grasping the other side of the limb, the thin flap is shaved off with a razor and immediately transferred to the denuded space. It may be slid directly from the razor by means of a fine probe and carefully adapted to the raw surface, which is to be completely covered either by a single flap or by several strips placed closely side by side. These flaps are applied to fresh wounds or to granulating surfaces. In the latter case Thiersch first scrapes off the superficial layer of the granulations with a sharp curette, as he considers new granulations, with their delicate capillaries prone to bleed, unfavorable to the adhesion of grafts.

OPERATIONS FOR PTOSIS.

As the levator in nearly all cases has little or no power, not much is to be expected from its advancement, and attempts in this direction have not proved successful. It may be considered applicable only to cases in which the muscle has been detached from the cartilage by a wound. In all other cases, except the hypertrophic, the operator's aim is to promote the supplementary action of the occipito-frontalis, and the result must at best be imperfect, as this muscle draws the lid up like a vertical curtain instead

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[1] Panas, Traité des Maladies des Yeux, t. ii. p. 176, from Beiträge zur Klin. Chir., 1889, Bd. iv. S. 625.

[2] Berlin. Klin. Wochenschr., 1874, S. 353, and Fifteenth German Surgical Congress, 1886. See, also, Jaesche, Klinische Monatsbl. f. Augenheilk., 1881, S. 40.

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of sweeping it upward and backward over the convexity of the ball, as is done by the levator.

The simplest operation is the removal of an elliptical piece of skin from the lid and bringing the edges of the wound together with sutures. This flap may be made as large as is possible without preventing closure of the eye. A slight apparent excess may, however, be allowed, as it will be overcome by the action of the orbicularis stretching the skin. This operation gives entirely satisfactory results when there is simple redundancy of the integument, and will often answer as well as any other in the slighter cases from other causes; but in the higher degrees of ptosis, particularly of the paralytic form, the effect is insufficient, and the deformity produced by the vertical stretching of the skin, with obliteration of the tarso-orbital fold, is a serious objection.

By Graefe's operation[1] the lid is shortened subcutaneously and the power of the orbicularis is weakened. A horizontal incision of the skin is made throughout the whole extent of the lid and five millimetres above its free margin, and the edges of the wound are undermined and separated widely by drawing them apart. A band of muscular fibres, eight or ten millimetres wide, is then excised down to the tarsus and tarso-orbital fascia, and the wound is closed by deep sutures that include the whole thickness of the muscle, as well as the edge of the skin. If the latter is redundant a strip of it may be excised.

Gillet de Grandmont[2] makes a subcutaneous excision of the cartilage as well as of the muscle. The extent of the excision required is determined by comparison of the distance between the free margin of the lid and the upper edge of the eyebrow on the two sides while the eyes are fixed on an object directly in front. The lid is held in a Snellen clamp with an ebony or horn plate, an incision of the skin two and one-half centimetres long is made parallel to the free lid-margin and three or four millimetres above it, the skin is undermined and its edges are stretched well apart, and the muscle is excised so as to lay bare nearly the whole of the cartilage. An incision two centimetres in length and from two to four millimetres above the lid-margin is then carried through the cartilage down to the plate of the clamp. The ends of this incision are united by a convex incision upward, whose height at the middle is determined by the amount of shortening required, and the semilunar piece of cartilage thus marked out is removed. The deep parts of the wound are united by three antiseptic catgut threads, not passed through the skin, which is allowed to close over the sutures.

Dransart[3] first proposed to connect the cartilage with the frontal muscle by means of subcutaneous cicatricial bands. He made an incision through the skin along the upper border of the tarsus and passed three

[1] Arch. für Ophth., ix. 2, p. 59.
[2] Journ. de Méd. de Paris, 1891.
[3] Annales d'Oculistique, July, 1880.

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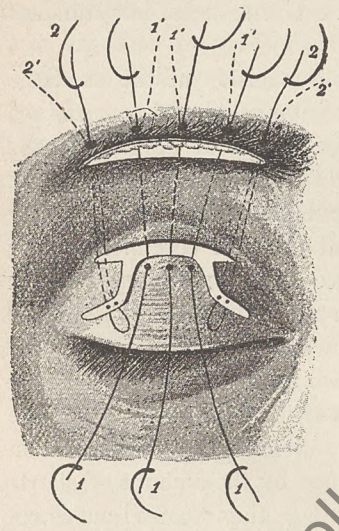
catgut ligatures, six or eight millimetres apart, through the cartilage and muscle to emerge above the eyebrow, knotted their ends, and allowed the skin to unite over them.

Pagenstecher[1] modified this operation as follows. The skin is not incised. Each end of a strong silk thread is attached to a needle; one needle is entered just above the lid-margin, passed horizontally beneath the skin, brought out two millimetres from entrance, re-entered at the same point and passed beneath the skin to emerge a finger's breadth above the eyebrow; the other needle is entered where the first one was, and passed directly up beneath the skin to emerge on the brow by the side of the point of exit of the first. The two ends of the thread are then tied over a piece of rubber drainage-tube or a roll of plaster, and it is either removed after some days, if sufficient inflammatory action has been excited, or is gradually drawn out above as the loop ulcerates its way through the subcutaneous tissue. Two such sutures are used.

Wecker[2] combines the operations of Graefe and of Pagenstecher. After excising a band of muscle, and in some cases a small flap of skin, as in the Graefe operation, he passes the two ends of the thread through the skin and muscle at the lower margin of the wound, leaving a bridge five or six millimetres wide between the entrance points, and beneath the muscle at the upper margin, brings them out on the brow, and ties them in a bow-knot over a roll of kid. The threads are tightened from time to time until the loop is drawn through as in the Pagenstecher operation. The result is the production of submuscular cicatricial bands and a depressed cicatrix of the skin in the natural fold of the lid.

Fig. 47.



Kunn[3] frees the insertion of the occipito-frontalis and unites it subcutaneously with the upper margin of the tarsal cartilage.

Panas[4] makes a horizontal incision two centimetres long down to the periosteum, just below the margin of the orbit, and one three centimetres long just above the eyebrow, and dissects up the bridge of skin and muscle between them. A tongue-shaped flap of skin and muscle is then formed on the lid, with its free end at the lower of these incisions and its base at the tarso-orbital fold, and is drawn up under the bridge to be stitched by three sutures to the upper edge of

[1] International Congress, London, 1881.
[2] Annales d'Oculistique, July, 1882, p. 29.
[3] Wien. Med. Wochenschr., 1893, 8 und 9.
[4] Arch. d'Ophtalmol., 1886, p. 1.

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the upper wound. This flap is eight millimetres wide, and at its base the incision is carried through the tarso-orbital fold at each side for the whole width of the lid. To prevent eversion of the lid-margin, two lateral sutures are used, including only the suspensory ligament and conjunctiva. The positions of these sutures are shown by the numbers in Fig. 47.

CANTHOPLASTY.

The object of this operation is to enlarge the contracted commissure by freeing the external canthus. It is frequently sufficient to incise the canthus as far as the margin of the orbit and unite the cut edges of the skin and conjunctiva with sutures. The lids are held apart and somewhat stretched by the fingers or a spring speculum, while the cut is made with a pair of strong scissors or with a grooved director and bistoury. This incision divides the skin, muscle, conjunctiva, and external palpebral ligament. After checking the hemorrhage by pressure or hot water, or by twisting one or two small arteries,—a ligature is rarely necessary,—the cut edge of the conjunctiva is seized with the forceps and sutured to the cut edge of the skin. Three sutures are usually sufficient,—one at each margin of the wound and one at the angle.

If necessary, a greater effect can be produced by cutting the palpebral ligament more freely, as recommended by the late Dr. Cornelius Agnew.[1] After making the incision described above, the palpebral ligament is made tense by drawing the upper lid towards the nose, when its cut edge can be felt at the upper border of the wound. The points of a small pair of scissors are inserted upward for four or five millimetres, one between the ligament and the skin and the other beneath the ligament, and the latter is incised. The lid will be felt to yield as the cut is made.

The method of Dr. David Prince[2] was proposed particularly for cases of contraction of the commissure accompanying entropion and as a part of the operation for the latter, but will often be found useful in other cases where there is great shrinking of the conjunctiva, or where a previous operation has left the tissues at the canthus in a cicatricial condition. From a point just outside the canthus carry an incision downward and inward from one-third to one-half the length of the lower lid and parallel to its free margin; and from a point on the same horizontal line with the first and five or six millimetres farther out, make another incision also downward and outward, to meet the first incision at its extremity. Dissect up the triangular flap of skin thus marked out to its base, and cut through the other tissues at the canthus by a horizontal incision beneath the flap. Undermine the upper edge of the wound, so as to free the outer angle of the lid. Pass a suture armed with a needle at each end through the point of the flap, introduce the needles under the upper edge of the wound, bring

[1] Annales d'Oculistique, 1875, 2, p. 186.
[2] Am. Jour. Med. Sciences, October, 1866, p. 381.

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them out through the skin beneath the brow, and tie the two ends of the thread over a roll of buckskin or plaster. The flap is thus doubled upon itself and presents a surface of sound skin to the lower edge of the wound. The suture is left four or five days, until the flap becomes united in its new position. The wound beneath the lid is closed by undermining its edges and uniting them with sutures. If there is no entropion, the flap may be made shorter and may be taken from a position more directly downward.

TARSORRAPHY.

Tarsorraphy is performed to diminish the extent of the palpebral commissure by contracting the canthus, or to keep the eye closed by uniting the lid-margins.

The Walther-Graefe operation is performed as follows. The commissure is pinched between the finger and thumb to determine the amount of contraction required, and the position of the new canthus is marked with a pen or the point of a knife. A horn spatula is held beneath the outer angles of the lids, and a strip of each lid-margin, about two millimetres wide to include the cilia bulbs, is excised by two cuts which meet beyond the canthus, and the fresh edges are brought together by sutures. The eye is covered with an antiseptic dressing, and the sutures are allowed to remain until firm union has taken place.

Fuchs splits the lower lid at the canthus by an incision separating the skin from the cartilage, and makes a perpendicular cut at the end of this incision to allow the anterior layer, containing the cilia, to be everted and the groove to gape. He then splits the margin of the upper lid in the same way, excises the anterior lip of the wound containing the cilia bulbs, and inserts the posterior lip consisting of cartilage into the groove in the lower lid, where it is held by sutures.

Wecker prefers to spare the cilia, and removes only the epithelial layer at the posterior edge of the lid-margin.

When the object is merely to protect the cornea, as in case of paralysis of the orbicularis or exophthalmos, it will be necessary only to unite the lids near their middle for a space of five or six millimetres, on either side of which useful vision may be retained. A more extensive ankyloblepharon may be required in operations for ectropion, but should never include the canthus if it is probable that a future reopening of the commissure may be desired. A thin slice of the inner edge of the opposed lid-margins is shaved off, along the line of the Meibomian orifices, and the raw surfaces are brought together by sutures. It is important that the threads should be introduced beyond the bared surfaces and take a good hold of the edge of the lid, as if only the anterior margins are included these surfaces are not held in contact. A thread with two needles introduced several millimetres beyond the lid-margins and not including the latter will bring the bared surfaces together still better by slightly everting the cilia.

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ANKYLOBLEPHARON.

Ankyloblepharon is usually accompanied with symblepharon. When there is adhesion of the margins of the lids only, without adhesion also to the ball, it is easily cured by a simple incision and frequent separation of the lids if the canthus is not involved. In the latter case there is likely to be more or less reunion of the divided edges, which may be prevented by performing a canthoplasty.

SYMBLEPHARON.

Symblepharon is a very difficult condition to treat, as is shown by the great number of operations that have been proposed. This difficulty is in proportion to the extent and closeness of the adhesions. When the lid is adherent to the ball only anteriorly, near its margin, and a probe can be passed along the cul-de-sac beneath the adhesion, it may be possible to prevent reunion after separation by a simple incision, but when the cul-de-sac also has been involved and there is a continuous raw surface extending from the lid to the ball, the symblepharon will be reproduced during the process of cicatrization in spite of all the ingenious procedures that have been suggested to prevent it. If the adhesion is comparatively narrow, and has been stretched into a band by the constant movements of the ball, it can be most satisfactorily treated by the method proposed by Arlt. The band is seized by the forceps and drawn forward, while it is detached from the ball with a small pair of scissors and dissected free well down to the cul-de-sac (lower lid). A thread with a needle at each end is passed through the free end of the flap, which is doubled upon itself. The needles are inserted into the bottom of the wound and brought out through the skin just above the margin of the orbit. The two ends of the thread are then tied over a small roll of plaster or buckskin, and the flap is held with its raw surface in contact with the raw surface of the lid and its sound surface opposed to the ball. The bared space on the ball is covered by uniting the edges of the divided conjunctiva, which, if necessary, may be undermined and freed by a horizontal incision at each side along the angle of the fold.

Closer adhesions, not involving the whole extent of the lid, may be treated successfully if the raw surfaces on the ball and lid, which lie in contact after the separation of the parts by incision, can be covered by sound tissue. The covering of even one of these surfaces may answer, if complete and carried well down to the bottom of the cul-de-sac. The best way of accomplishing this, if practicable, is by means of conjunctival flaps. This method has been particularly advocated by Teale,[1] who, after freeing the lid from the ball, takes a large flap from the conjunctiva on each side of the cornea, and covers the bared space on the ball with one and that on the lid with the other. In separating the lid from the ball, he makes the incision along the corneal margin and leaves the portion adherent to the

[1] Ophthalmic Hospital Reports, vol. iii. p. 270.

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cornea, to avoid adhesion of the conjunctiva and cicatricial dragging at this point. This patch is subsequently to a great extent absorbed. Later,[1] Teale restored the conjunctiva of the lower part of the ball by a flap transferred from the upper part. A band of conjunctiva of sufficient width is included between two incisions parallel to the corneal margin, dissected free except at the ends, which are left detached, brought down over the cornea, and sutured in its new position.

Transplantation of mucous membrane from the lip and from the vagina, of skin-flaps without pedicle, of the rabbit's conjunctiva, and of the skin of the frog has been tried with indifferent success.

Recently the Thiersch graft has been used to replace the conjunctiva with very satisfactory results.[2] After the separation of the lid and ball a graft may be applied to each in the usual way. The eye is closed for several days with an antiseptic dressing. The graft in successful cases adheres firmly, and subsequently contracts considerably, but remains opaque and whitish and never assumes the appearance of conjunctiva. This method has thus far been used only in partial symblepharon, but may have an application in cases of more extensive adhesion. It has the advantage over skin-flaps that it can be more readily applied to the ball. It also has a firm base on the ball, while when it is transferred to the dissected lid the inodular tissue upon which it rests is subject to a high degree of cicatricial action.

Close adhesion of the whole surface of the lid to the ball, without cul-de-sac or canthus, is a most discouraging condition to treat. Fuchs, in his recent text-book, states definitely that cases of extensive symblepharon in which the adhesion reaches the fornix are incurable, and most surgeons will at least agree with Panas, that its satisfactory treatment "*reste encore à trouver.*"[3] Efforts continue to be made, however, and cases are reported in which a partial cure has resulted. The fact that skin when placed in the position of conjunctiva assumes a character closely resembling that of mucous membrane has encouraged attempts to use it in operations for symblepharon.

Samelsohn[4] transplants skin from the upper lid to the inner surface of the lower, or from the lower lid to that of the upper. After completely separating the adhesion (lower lid), he dissects a quadrilateral flap from the upper lid, leaving its base attached at the ciliary margin, turns it down, applies its raw surface to the inner surface of the liberated lower lid, and sutures its free edge at the bottom of the cul-de-sac. The epithelial surface of the flap is thus opposed to the ball. The eye is left covered by an antiseptic dressing for five days to give time for union to take place, when the base of the flap is cut along the margin of the lower lid.

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[1] Fourth Internat. Ophthal. Congress, 1872.
[2] Annals of Ophthalmology and Otology, April, 1893.
[3] Arch. d'Ophtalmol., November, 1891.
[4] Annales d'Oculistique, Août, 1892, p. 139.

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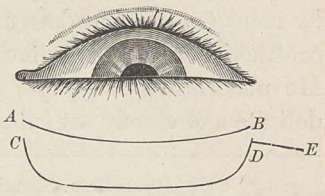
In the "Transactions of the American Ophthalmological Society" for 1890 I have reported an operation for extensive symblepharon, in which the liberated lower lid was lined with skin taken from the cheek. The lid was freely dissected from the ball until there was no limitation of the upward movement of the latter. An incision *AB* (Fig. 48) just above the margin of the orbit was carried through the whole thickness of the lid, which was separated completely from its attachments except at either extremity. A thin flap *CD* a little wider than the lid was then dissected from the skin just below it, turned up, applied with its raw surface to the inner surface of the lid, and held in place by suturing its free edge to the lid-margin. This left a deep cul-de-sac with sound skin turned towards the ball. Care was taken in dissecting up the flap not to separate it at its base-line, and to carry the incisions a little deeper there into the muscle, to allow it to be inverted without strain. The bared space on the cheek was covered by undermining the edges of the wound and forming a sliding flap. The immediate result was perfect, but some months later the cul-de-sac was to a great extent obliterated by cicatricial contraction, and there was adhesion to the ball at the inner angle of the lid, where the union of the flap had been imperfect. There was, however, very great improvement, with comparative freedom in the movements of the ball. A difficulty encountered in the treatment was the interference with healing caused by the tears, which resulted in a fistula at each end of the wound. This difficulty would be escaped in the case of the upper lid.

FIG 48.



OPERATIONS FOR EPICANTHUS.

In operations for epicanthus, either an elliptical piece of skin is removed from the bridge of the nose, or the redundant folds are attacked directly. The former method was first practised by Von Ammon under the name of rhinorraphy.[1] He raised with the forefinger and thumb a vertical fold of skin sufficient to cause the disappearance of the deformity and marked out its base with a pen, excised the elliptical flap thus outlined, and brought the edges of the wound together with harelip sutures.

Wecker,[2] after pinching up the fold, passes threaded needles through its base and excises it in front of them with curved scissors, and then, drawing the needles through, ties the threads.

It has been recommended to leave the wound to close by granulation to secure greater contraction, but this practice promotes, what it should be the operator's greatest care to avoid, a disfiguring vertical cicatrix.

The elder Graefe[3] raised the fold of the epicanthus with a pair of

[1] Zeitschrift f. Ophthalmol., Bd. i. S. 533, 1831.
[2] Wecker et Landolt, t. i. p. 180.
[3] Schon, Zeitschrift f. Ophthalmol., Bd. ii. S. 120.

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forceps, excised a triangular piece of it with the scissors, cutting deeply into the underlying tissue, and left the wound to granulate.

Arlt[1] excised a rhomboidal piece of the fold of the epicanthus with its long axis vertical, and sutured the wound horizontally.

Knapp[2] finds that operations upon the fold are likely to be followed by recurrence of the deformity, due to puckering of the skin by cicatricial contraction of the subcutaneous tissue, and prefers Von Ammon's method. He undermines the edges of the wound freely, and unites them with delicate and closely set sutures, instead of with a harelip pin.

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[1] Graefe u. Saemisch, Bd. iii. S. 443.

[2] Archives of Ophthalmology and Otology, vol. iii. p. 48.

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DISEASES OF THE LACRYMAL APPARATUS.

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THE lacrymal apparatus is divisible into two distinct parts: the one, consisting of the lacrymal gland and its ducts, has to do with the secretion of the tears and their carriage to the conjunctival sac; the other, comprising the puncta, the canaliculi, the lacrymal sac, and the nasal duct, is known as the drainage apparatus of the eye, and its function is to carry away the tears after they have performed their office of keeping moist and washing free of foreign particles the conjunctival sac and cornea.

ANATOMY.

The *lacrymal gland*, which is of the acinous variety, consists of two portions. The larger portion, known also as the superior lacrymal gland, lies in a depression in the roof of the orbit, the fossa glandulæ lacrymalis, just within the upper outer orbital margin. It is meniscoid in shape, and is about twenty millimetres long, twelve millimetres wide, and four millimetres thick. Its convex surface is in contact with the periosteum of the orbital roof, to which it is attached by fibrous bands. Its concave surface is in apposition with the upper and outer portion of the eyeball and with the superior and external recti muscles. The smaller division of the gland—the inferior or accessory lacrymal gland—consists of only one or two lobules, which lie along the excretory ducts of the superior gland, just beneath the mucous membrane of the fornix. The upper lid being everted, and the eyeball directed downward, the location and form of this portion of the gland may sometimes be well seen. In its structure and its general appearance the lacrymal gland resembles the salivary glands. The ducts which carry the tears from the two portions of the gland to the conjunctival sac vary in number from six to ten or twelve. They run obliquely for a short distance beneath the mucous membrane, and open by a series of minute orifices into the outer part of the upper conjunctival fornix.

In this connection mention should be made of the glands of Krause, as they are supposed to assist in the lacrymal function. They are small

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round or oval bodies, identical in structure with the lacrymal gland, and situated mainly in the superior conjunctival cul-de-sac, a smaller number being found in the inferior cul-de-sac.

Under ordinary conditions the lacrymal gland and the glands of Krause, just described, secrete only a sufficient quantity of tears to keep the conjunctival sac and the cornea moist and to assist in moistening the mucous membrane of the nasal passages. It is asserted that it is only the palpebral portion of the lacrymal gland which acts under such circumstances. Under psychical stimulation, or in consequence of irritation or inflammation of the eye, the superior and major portion of the gland becomes active, and the flow of tears is greatly increased, so that they run over the lids upon the cheek, and are discharged into the inferior nasal meatus in such quantity as to necessitate frequent blowing of the nose. In exceptional instances the activity of the gland is in a measure under the control of the will, it being possible to bring about a flow of tears whenever the individual may so will. The tears consist of little else than water and chloride of sodium, the solid constituents, including epithelium, albumin, chloride of sodium, alkaline and earthy phosphates, fats, and extractive matter, amounting to less than one per cent.

The *drainage apparatus* begins with the lacrymal puncta, two small openings situated upon the free margin of the lids, near the inner extremity of the tarsal cartilages. They are situated upon small elevations, the lacrymal papillæ, and face towards and lie in contact with the conjunctival surface of the eyeball. They are known respectively as the upper and the lower punctum, and they constitute the orifices of the little canals which run from this point to the lacrymal sac, the canaliculi lacrymales. (Fig. 1.) These minute canals are situated just beneath the free margin of the lids. Their course upon leaving the puncta is at first vertical; they then turn abruptly and run in a horizontal direction towards the lacrymal sac. As they approach the sac they converge, and they frequently blend into a common duct before the sac is reached; but as frequently this blending does not occur, and they pass into the sac as separate canals.

The lacrymal sac, the walls of which consist of a fibrous, elastic coat, lined with cylindrical epithelium, lies close to the inner canthus, and is lodged in a groove formed by the lacrymal bone and the nasal process of the superior maxillary. It is ovoid in shape, its upper extremity being closed in and rounded, while below it is continuous with the lacrymal duct. The internal palpebral ligament passes across and is closely connected with its anterior wall. (Fig. 1.)

The line of demarcation between the lacrymal sac and the lacrymal duct is not one that can be sharply drawn. Most authorities, in their description of the lacrymal passages, are rather nebulous upon this point. Fuchs says, "At the spot where the cleft of the lacrymal bone merges into the bony canal the lacrymal sac passes into the nasal duct," and he adds, "the point where this transition occurs constitutes the narrowest part of

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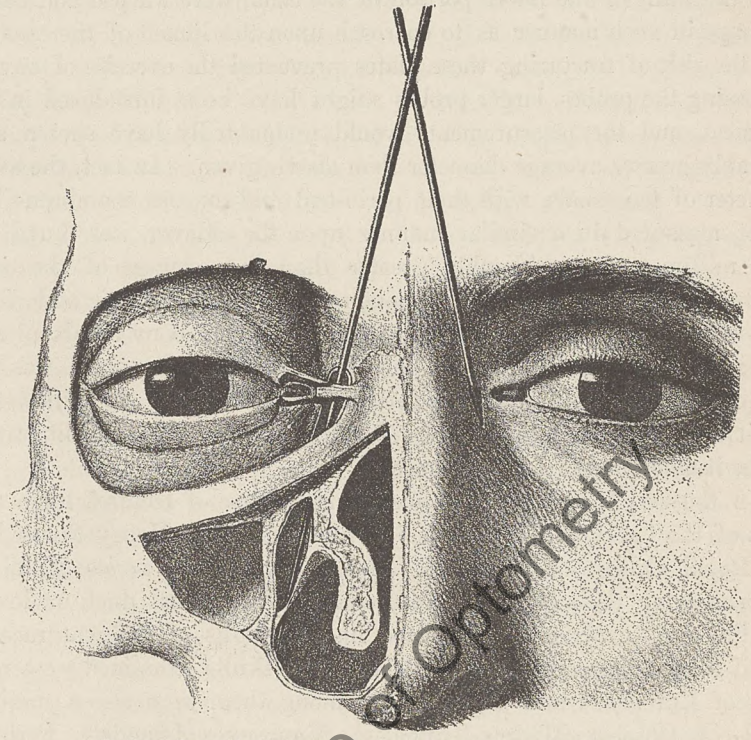
the whole lacrymal channel, and is, therefore, particularly liable to the formation of pathological contractions."[1] The lacrymal or nasal duct, which is about three-quarters of an inch in length, extends from the lower extremity of the lacrymal sac to the inferior meatus of the nose, its direction (which varies considerably in different individuals) being downward, outward, and slightly backward. Its nasal orifice, which varies in shape from an oval to a simple slit, has a valve-like formation, and is situated upon the lateral wall of the nose, beneath the inferior turbinated body. The duct is lodged in an osseous canal formed by the superior maxillary,

FIG. 1.



Section of canaliculi, lacrymal sac, and nasal duct. (De Wecker.)

the lacrymal, and the inferior turbinated bones. Its walls, like those of the middle ear, partake of the character of both a mucous and a periosteal membrane, and are supplied with a dense plexus of veins, resembling the venous plexuses of the turbinated bodies. Like the lacrymal sac, it is lined with cylindrical epithelium. The mucous membrane lining the duct, which contains numerous acinous mucous glands, forms at certain points valve-like folds, which encroach somewhat upon the lumen of the canal.

The bony canal which lodges the membranous lacrymal duct varies greatly both in size and in shape, and even in the same individual marked differences in these respects may exist between the canals of the two sides.

[1] Text-Book of Ophthalmology, English translation, p. 509.

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A cross-section at the upper extremity of the canal is usually oval in shape, with the long axis running from outward and forward backward and inward, but may be almost or quite circular. A series of measurements which I made some years since[1] showed that in exceptional instances the bony canal is so large as to permit the passage of a rigid probe having a diameter of seven millimetres, while in other exceptional instances a probe of only three millimetres' diameter can be passed. The average diameter of seventy adult canals measured in this way (by the passage of rigid probes) was 4.11 millimetres. But for the fact that in the skulls upon which these measurements were made the thin plates of bone which in part form the walls of the lower portion of the canal were warped and bent out of shape in such manner as to encroach upon the lumen of the canal, and that the risk of fracturing these plates prevented the exercise of any force in passing the probes, larger probes might have been introduced in many instances, and the measurements would undoubtedly have shown a considerably greater average diameter than above given. In fact, the average diameter of ten canals, with their periosteal and mucous membrane lining intact, measured in a similar manner upon the cadaver, was found to be 4.47 millimetres, considerably greater than the average of the osseous canals, because here the plates of bone referred to were elastic and yielding and occupied their normal position. In the seventy bony canals an appreciable difference in the size of the canals of the same skull was noted eighteen times. In four skulls this difference was only .25 millimetre, in ten it was .50 millimetre, in one .75 millimetre, in two 1 millimetre, and in one it amounted to 1.25 millimetres.

In the very interesting and instructive course of lectures upon "Diseases of the Lacrymal Apparatus," delivered by Mr. Henry Power before the Royal College of Surgeons, London, in 1886,[2] a series of tables is given showing the results of measurements of the nasal duct, made under his direction, in about five hundred human skulls of different races contained in the museum of the College. The skulls examined were mostly those of Europeans and negroes, but among them were also a number of skulls of Chinese, Madras, Cingalese, Vancouver Islanders, Peruvians, Polynesians, and Andaman Islanders. The tables show that the length and calibre of the lacrymal duct vary markedly in different races. The duct was found to be longest in the skulls of Europeans and of Peruvians, the average length from the floor of the orbit to the floor of the nose in two hundred and ninety-two European skulls being 27.47 millimetres. The average length in the negro skull was 26.1 millimetres, and in those of the Andaman Islanders only 22.4 millimetres. On the other hand, the calibre was least in the European and greatest in the negro and Chinese skulls. Separate measurements were made of the antero-posterior and of the lateral

[1] The Use of Large Probes in the Treatment of Strictures of the Nasal Duct, Transactions of the Medical and Chirurgical Faculty of Maryland, 1877, p. 154.

[2] London Lancet, 1886, vol. ii.

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diameters of the ducts, the latter being the shorter. The average of the lateral diameters of two hundred and five ducts in European skulls was 3.77 millimetres, while that of one hundred and eighty-one ducts in negro skulls was 4.7 millimetres, and of thirty-six ducts in Chinese skulls 4.63 millimetres.

In my own measurements no effort was made to identify the race to which the skulls belonged, but it is probable that many of the skulls examined were those of negroes. If we take the mean between the average lateral (least) diameter of the European and that of the negro skulls, as given by Mr. Power, and compare it with my results, we shall find, although the measurements were made by different methods, that there is a pretty close agreement between the two, the mean of his measurements being 4.23 millimetres, and of mine, as already stated, 4.11 millimetres.

Many ingenious theories have been advanced to explain the perfect way in which, under normal conditions, the tears are drained from the conjunctival sac and carried to the floor of the nose. The act of winking is certainly an important factor in bringing about this result. That the winking of the lids does, as a matter of fact, facilitate the disappearance of tears from the eyes, especially when they are there in inconveniently large quantity, is probably within the experience of every one. It seems probable that the winking has a twofold action in accomplishing this result. In the first place, it unquestionably carries the tears from the general conjunctival surface of the eye towards the caruncle, causing them to collect in the neighborhood of the lacrymal puncta; in the second place, it is probable that the pull which the orbicularis muscle, in contracting, exerts upon the internal palpebral ligament is, through the intimate connection existing between the latter and the anterior wall of the lacrymal sac, extended to the sac itself, and that the effect of this is to produce a sort of suction-pump action, which draws the tears through the puncta and the canaliculi into the sac. Besides this action of the lids, it would seem that when the tears reach the puncta they must be drawn into the canaliculi, in some measure at least, simply through the influence of capillary attraction. Once having entered the lacrymal sac, they descend through the duct to the nose as a result of their own gravity. It is believed, however, that this descent is facilitated by the elasticity of the lacrymal sac, which causes it when distended by tears to contract upon its contents.

DISEASES OF THE LACRYMAL GLAND.

Diseases of the secretory portion of the lacrymal apparatus are, comparatively speaking, of rare occurrence. This fact is explained in a measure by the protected position which the gland occupies and by the multiple system of ducts with which it is provided. The immunity which the gland enjoys from involvement in the more virulent conjunctival inflammations, such as gonorrhœal ophthalmia, is worthy of special remark.

The lacrymal gland is occasionally the seat of inflammation (dacryo-

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adenitis), which may be of acute or chronic character; neuralgia of the gland (dacryo-adenalgia) is recognized; hypertrophy and atrophy of the gland are met with; new growths sometimes select it for their site; cystoid dilatation of one or more of its ducts (dacryops) occurs; chalky concretions (dacryoliths) may form in it; it has been known to undergo dislocation, both traumatic and spontaneous; and its inflammation or injury may give rise to the formation of a persistent variety of lacrymal fistula.

Dacryo-adenalgia is the name proposed by A. Schmidt for neuralgia of the lacrymal gland, which he was the first to describe. It is characterized by severe pain in the region of the gland, by photophobia, and by excessive lacrymation. It is said to occur especially in children, in pregnant women, and in the subjects of gout. It may assume a chronic character, and is prone to recur. It is probably a rare affection anywhere, and is certainly so in the United States. The local remedies which suggest themselves for its relief are atropine in the form of a collyrium, the application to the brow and over the region of the gland of oleate of morphine, or oleate of morphine and atropine, moist or dry heat, and the liberal use of a lotion of opium (ext. opii, gr. x to xv; aquæ, oz. iv) or of belladonna (of similar strength). Quinine, iron, and other suitable constitutional remedies should also be administered.

Hypertrophy of the lacrymal gland is said to occur especially in children, and has been known to be of congenital origin. It may reach such proportions as to force the eye from the orbit and destroy sight through stretching and compression of the optic nerve. The accompanying illustration (Fig. 2) represents faithfully a remarkable case of simple hypertrophy of the lacrymal gland which came under the observation of the late Professor Christopher Johnston, of Baltimore, in 1876. Although the eye was forced so far from its normal position in the orbit, it still retained its movements, and possessed a visual acuteness equal, at least, to counting fingers. The gland, which was about the size of a hen's egg, and was found to contain numerous dacryoliths, composed of concentric layers of carbonate of lime, was removed by Professor Johnston through an incision made parallel with the orbital margin. The eye, in time, resumed nearly its normal position, but binocular vision was not regained. When the enlargement of the gland is marked, and vision is suffering in consequence, enucleation of the hypertrophied gland should be resorted to without delay; but, when this is not the case, the local application of iodine or mercury, electrolysis, and the internal administration of the iodides may be tried, in the hope that the hypertrophic process may be arrested.

Atrophy of the lacrymal gland occurs only very rarely. It has been met with in the advanced stage of xerosis of the conjunctiva, or xerophthalmia. Arlt has described a case of this character in which, as a result of the shrinking of the conjunctiva, the efferent ducts of the lacrymal gland were obliterated, and the gland itself was reduced to one-third of its normal size and was transformed into a tissue resembling fat. In paralysis

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FIG. 2.

Hypertrophy of the lacrymal gland, producing marked exophthalmus.

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of the trigeminus, in consequence of the innervation of the lacrymal gland being disturbed, abolition of the lacrymal secretion may occur. (Fuchs.)

Harlan, of Philadelphia, has reported an interesting case[1] in which a servant-girl, twenty-two years of age, received a severe burn of the left side of the head and face, which caused ectropion of both lids and obliteration of the lower punctum, the position of the upper punctum being such as to render it incapable of performing its office. Although the normal moisture of the conjunctiva was preserved, there was no epiphora, and when the patient cried, no tears were secreted by the lacrymal gland of this eye. Harlan thought it improbable that the gland had been destroyed by the burn, and was inclined to attribute the suppression of the lacrymal secretion to the obliteration and eversion of the puncta. He also refers to another case in which, in removing a tumor from the neighborhood of the inner canthus, he necessarily destroyed both puncta. Several months after the operation the patient, much to her surprise, discovered that she could cry only with the opposite eye. In other respects she noticed no difference between the eyes. In this connection he refers to the observation that artificial destruction of the lacrymal sac is at times followed by a similar suppression of the function of the corresponding lacrymal gland. A case of "congenital unilateral absence of lacrimation,"[2] thought to be due to absence of the lacrymal gland, has been reported by Mr. A. Stamford Morton.

Dacryo-adenitis, or inflammation of the lacrymal gland, occurs under two forms, as a chronic and as an acute affection. Both varieties are rare; the latter variety very rare. Arlt, in his "Lehrbuch" (1853), says that he had never seen a case of dacryo-adenitis, and Hirschberg states[3] that among twenty-two thousand five hundred recorded cases of diseases of the eye he had met with but one case of suppurative inflammation of the gland. Power asserts[4] that the indices of the Royal London Ophthalmic Hospital Reports make mention of only one case of abscess of the lacrymal gland. It seems probable, however, that the disease is not so extremely rare as might be inferred from these data, for its diagnosis is not always an easy matter, and it has, without doubt, often been mistaken for simple orbital cellulitis. It is said to occur more frequently in children than in adults, and oftener in women than in men. (Rooley.) Galezowski has called attention to the fact that dacryo-adenitis sometimes assumes an epidemic character, and he states that he once met with an unusual number of cases during an epidemic of mumps. Dr. C. E. Rider, of Rochester, New York, has also reported two cases of inflammation of the lacrymal gland occurring in connection with mumps.[5]

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[1] Philadelphia Medical Times, 1871–72, vol. ii. p. 124.

[2] Trans. of the Ophthalmological Society of the United Kingdom, vol. iv. p. 350.

[3] Archives of Ophthalmology, vol. viii. p. 369.

[4] London Lancet, July 31, 1886.

[5] Trans. Med. Society of New York, 1872, p. 158.

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The chronic variety of dacryo-adenitis is characterized by an enlargement of the gland, which may usually be recognized by palpation, by swelling and redness of the upper lid, especially over the region of the gland, and by injection of the conjunctiva, most marked in the neighborhood of the superior retrotarsal fold. Pain is not a prominent symptom, but the gland is usually sensitive to pressure. When the upper lid is everted and the eye directed downward, the lower portion of the swollen gland comes into view as a red, tongue-shaped, nodular mass. (Hirschberg.) If the swelling of the gland be considerable, displacement of the eyeball downward and inward, with impairment of its mobility, may occur, and this will be attended by diplopia.

In acute inflammation of the gland all the symptoms just enumerated are present in exaggerated form, and are accompanied by pain of severe character, elevation of temperature, and in some instances cerebral excitement, sleeplessness, and delirium. There is not only injection but also marked chemosis of the conjunctiva, which, especially from above, may overlap and hide from view the cornea. The œdema and swelling of the lids are so great as to suggest the appearance which characterizes purulent ophthalmia. (S. C. Ayres.) The eyeball may be greatly displaced by the enlargement of the gland and its movements restricted and accompanied by pain. The great œdema of the lids makes it difficult to palpate the gland, or to bring it into view, as may be done in cases of chronic dacryo-adenitis. Suppuration may supervene within a few days, or, as very often happens, the inflammation may subside without the formation of pus. When pus does form, it may make its way to the surface through the integument of the lid, or it may discharge into the superior conjunctival cul-de-sac. The affection is usually unilateral, but not infrequently both glands are simultaneously affected. Nettleship and others have met with cases of dacryo-adenitis in which the inflammation was confined to the inferior or accessory portion of the gland, the main part of the gland not being involved.[1]

Inflammation of the lacrymal gland has been ascribed to numerous causes; among them may be mentioned traumatism, "cold," rheumatism, gout, struma, syphilis, septic absorption, mumps, and the extension of inflammation from the conjunctiva and cornea. Dr. Pooley has reported an interesting case[2] in which a severe acute inflammation occurred in the lacrymal gland of one eye shortly after the loss of the other eye by diphtheritic conjunctivitis. The first eye was in the state of purulent inflammation when the dacryo-adenitis manifested itself in the opposite eye, and Dr. Pooley inclines to the belief that the adenitis was due to septic absorption. During the discussion of this case (in the American Ophthalmological Society), Dr. H. G. Miller mentioned a case of bilateral, acute inflammation of the lacrymal gland which he had met with in a patient who

[1] St. Thomas's Hospital Reports, 1877, N. S., vol. viii. p. 240.

[2] Trans. American Ophthalmological Society, vol. iii. p. 729.

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was suffering at the time with urethral gonorrhœa. Here also it seems probable that the adenitis was of septic origin, being of a like nature with the iritis and arthritis which occasionally manifest themselves during the course of an attack of gonorrhœa. Many authorities mention the fact that inflammation of the lacrymal gland is frequently found associated with chronic conjunctival and corneal inflammation. (Pooley.)

The treatment of dacryo-adenitis will vary with the nature of the attack and the causes which may have given rise to it. In the chronic variety, the local application of mercurial ointment, oleate of mercury, or compound iodine ointment will be found useful, while the iodide of mercury, iodide of potassium, salicylate of sodium or lithium, quinine, and iron are the constitutional remedies which are likely to prove of most value. Extirpation of the gland may become necessary, should it enlarge to such an extent as to endanger the integrity of the eye. In the acute form of the disease, leeching and the liberal application of an ointment of mercury and belladonna or opium (ext. opii vel ext. belladonnæ, dr. i; ung. hydrarg., oz. i), together with the administration of an energetic calomel cathartic, to be followed by free doses of quinine, salicylate of sodium, or pyrophosphate of sodium (the latter to be given in twenty-grain doses every two hours, to be effectual), if resorted to at the beginning of the attack, may, perhaps, result in cutting it short. Should this not prove to be the case, poultices or warm fomentations, to which opium or belladonna may be added, should be applied, and as soon as the presence of pus can be detected it should be evacuated by a free incision made either through the integument of the upper lid or from the conjunctival cul-de-sac, as may seem to be indicated by the tendency of the abscess to point. For the relief of pain, morphine or opium will be required; or, if the pain be not marked, sulphonal may be given instead.

As a sequela of inflammation of the lacrymal gland, or in consequence of traumatism, a troublesome and obstinate *lacrymal fistula* occasionally forms, and tears continue to be discharged through the opening in the integument of the lid which originally permitted the escape of pus or which may have resulted from the traumatism. Under such circumstances the opening fails to heal and the individual suffers much inconvenience. Congenital fistula of the lacrymal gland has also been observed. It is difficult to bring about a closure of such a fistula, and, moreover, if we succeed in accomplishing this we do so at the risk of precipitating a fresh attack of inflammation of the gland. The operative procedure which has proved most successful is that first suggested and practised by Sir William Bowman.[1] It consists, essentially, in converting a troublesome cutaneous fistula into one opening into the conjunctival sac and hence giving little or no annoyance. A threaded needle is passed a short distance into the fistulous passage and is then made to penetrate the lid, being brought out through

[1] London Ophthalmic Hospital Reports, vol. i.

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its conjunctival surface. A second needle, upon the opposite end of the same thread, is next passed through the lid, just at the external orifice of the fistula. The two ends of the thread are then tied tightly, so as to include and constrict a bridge of conjunctiva and lid integument, and are left to cut their way out. To promote the closure of the external orifice of the fistula, its edges should be freshened. Temporary fistulæ, of a different character, may occur in consequence of caries and necrosis of the margin and roof of the orbit dependent upon severe dacryo-adenitis. The healing of such fistulæ will, of course, follow the removal of the diseased bone.

Cyst of the lacrymal gland, known as *dacryops*, and due to obstruction of one or more of the efferent ducts of the gland, is occasionally met with. Upon eversion of the upper eyelid the cyst may be brought into view, and appears as "a bluish-pink, semi-transparent, elastic, and somewhat fluctuating swelling, consisting, perhaps, of several nodulated segments of varying size. . . . The swelling, moreover, increases suddenly and markedly in size if the patient cries or the secretion of tears is stimulated by the application of some irritant to the conjunctiva." (Soelberg Wells.) If the cyst be of considerable size it may be evident to the eye without eversion of the lid, and may also be detected by the touch. In some instances the occlusion of the efferent duct is incomplete, so that the cyst, after becoming distended during the activity of the gland, presently returns to its previous smaller size through the slow oozing out of the tears. Usually, in such cases, the cyst may be entirely emptied by pressure. Cysts of the lacrymal gland are occasionally congenital. It is impossible, owing to the delicate and ill-defined character of its walls, to dissect out a cyst of the lacrymal gland, and the treatment which has proved most successful consists in establishing an artificial opening between the cyst and the conjunctival sac. A linear incision may be made through the cyst-wall and kept patent by the daily introduction of a probe until its edges have cicatrized, or, as suggested by Von Graefe, a silk thread may be passed through the wall of the cyst, tied in a loose loop, and permitted to cut its way out.[1]

Dacryoliths, or chalky concretions, sometimes denominated *lacrymal calculi*, form, in rare instances, in the lacrymal gland. They are liable to cause mechanical irritation, and should be removed through an incision made from the conjunctiva.

Tumors of the Lacrymal Gland.—New growths having their origin in the lacrymal gland are rare. They usually occur in persons of advanced age, and occasionally are traceable to some previously received injury. The glands of both eyes have been known to be simultaneously involved. They are usually of slow growth, and are apt to be almost or quite immovable. As they increase in size they interfere with the movements of the eye, and

[1] An interesting paper upon Fistulæ and Cysts of the Lachrymal Gland, by Mr. Hulke, may be found in the Royal London Ophthalmic Hospital Reports, vol. i. p. 285.

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by pressure upon the ball cause a squint, which is usually downward and inward. Later, as they spread towards the apex of the orbit, they crowd the eyeball forward, giving rise to marked exophthalmos. Though they may surround the optic nerve, they scarcely ever invade it. Atrophy or inflammation of the nerve may, however, ensue as a result of the traction and pressure exerted upon it, and ulceration and sloughing of the cornea may later make their appearance in consequence of the exophthalmos and the disturbed nutrition of the eye. Through absorption of the orbital walls the tumor may enter the cerebral cavity, and by involving the brain-substance cause the death of the individual. (Alt.[1])

Alt states[2] that he has seen and examined the following varieties of tumors which, in his opinion, unquestionably had their origin in the lacrymal gland: adenoma, showing the typical picture of an epithelial neoplasm; myxoma and myxo-sarcoma, characterized by the development of spindle-cells with long offsets and stellate cells embedded in a mucoid intercellular substance, and by the gradual disappearance of the glandular structure; spindle-cell sarcoma,[3] consisting of densely packed spindle-cells in the older, and round cells and small spindle-cells in the younger portions of the growth; lympho-sarcoma, made up of densely packed lymphatic cells, with hardly any intercellular substance; and epithelioma, in which solid epithelial cell cylinders and nests of epithelial cells take the place of the gland-substance, this being usually accompanied by the development in the gland of myxomatous tissue. Knapp has reported one case each of myxo-adenoma, adenoma carcinomatosum, and myxo-adenoma carcinomatosum of the lacrymal gland,[4] and Fuchs mentions, among other growths which have been met with, carcinoma, cylindroma, lymphadenoma, chloroma, and sarcoma.[5] Hydatid cysts of the gland, dermoid growths, and angioma have also been described.

Complete removal of the new growth by operation is the only measure which is likely to prove of benefit. If the opportunity to do this presents itself before the growth has become too extensive, it is frequently possible to remove the diseased tissue without sacrificing the sight. On the other hand, when the tumor has reached a more advanced stage, and has surrounded the eyeball and involved the deeper parts of the orbit, enucleation of the eye becomes almost a necessary step in any operative procedure for the eradication of the disease.

Removal of the lacrymal gland may be accomplished by either of two procedures. The gland may be exposed by an incision through the integu-

[1] Reference Hand-Book of the Medical Sciences, vol. ii. p. 813.

[2] Ibid.

[3] A case reported by Alt in the American Journal of Ophthal., 1885, vol. ii. p. 201. Also a case of adenoma, Archives of Ophthal., 1880, vol. ix. p. 288.

[4] Trans. American Med. Assoc., 1880, vol. xxxi. p. 665.

[5] A case of sarcoma of the lacrymal gland has been reported by Harlan, Trans. American Ophthal. Soc., vol. iii. p. 402, and one of myxo-adeno-sarcoma by White, of Richmond, Archives of Ophthal., 1882, vol. xi. p. 62.

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ment of the lid, drawn out by means of a tenaculum, and with a knife or scissors separated from its attachments. The objection urged against this method is that it involves a more or less complete division of the levator palpebræ superioris, and so may result in the production of ptosis. The other and probably better plan, suggested by Velpeau, is to divide the external canthus, evert the upper lid, and cut down upon the gland through the conjunctiva, at the level of the oculo-palpebral fold. This plan does not endanger the integrity of the levator muscle, and leaves a less conspicuous scar than is left by the first-described procedure.

Dislocation of the lacrymal gland, as might be supposed from its protected position, happens only very rarely. Nevertheless, a few cases of traumatic luxation of the gland have been reported, and also a very few in which the luxation occurred without traumatism. Von Graefe met with a case of the former variety, in which he was able to restore the gland to its normal position,[1] and Rampoldi has described another, in which a blow with a stick upon the temporo-parietal region gave rise to a retrobulbar abscess, which was followed by the development of ectropion of the upper lid and luxation of the lacrymal gland.[2] In this instance the gland could not be replaced, and Rampoldi removed it by a procedure similar to that proposed by Borelli for the removal of staphyloma.

An interesting case of non-traumatic dislocation of the gland has been reported by Snell, of Sheffield.[3] It occurred in a man forty-five years of age. The gland could be seen and felt as a tumor about the size of an almond beneath the structures of the left upper lid. It could be easily replaced, but when the head was bent forward reappeared in its previous position. The dislocation first occurred during the night, there having been no antecedent traumatism. There was, however, a large nævus on the same side of the head, which involved the eyebrow and extended into the corresponding orbit. The gland was replaced by pressure with the fingers, and five weeks afterwards was found to be still in its normal position. Snell regarded the case as being unique. He mentions, however, having met with an instance in which, as a congenital defect, both lacrymal glands were found to be movable in the eyelids. A case of spontaneous dislocation of the lacrymal gland has also been recorded by Noyes. It occurred in a young girl, and is described as having been due to "slow relaxation of the enclosing capsule. The gland presented itself beneath the ocular conjunctiva, over the insertion of the rectus externus muscle, and was affected by a slight degree of inflammation, which was not, however, the cause of the displacement. The opposite eye was phthisical, and there, too, the degenerated and atrophied lachrymal gland had descended below its proper place."[4] Mauthner has reported a case of "prolapse" of

[1] Archiv f. Ophthalmol., Bd. xii. p. 224.
[2] Annali d'Ottal., an. xiii., fasc. 1.
[3] Ophthalmic Review, London, vol. i. p. 207.
[4] Diseases of the Eye, New York, William Wood & Co., 1890, p. 272.

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the lacrymal gland,[1] and Brière has described a case of "hernia" of the entire gland, accompanied by ectropion and marked elongation of the upper lid, consequent upon caries of the orbit.[2]

DISEASES OF THE DRAINAGE APPARATUS.

Although the secretory portion of the lacrymal apparatus, as has been indicated, is so rarely the seat of disease, quite the reverse is true of those structures which together constitute the lacrymal drainage apparatus. There is not one of these which is not liable to pathological changes. The puncta, the canaliculi, the lacrymal sac, and the nasal duct all are frequently the seat of disease processes. The puncta are liable to occlusion, or may assume a malposition which interferes with their function; the canaliculi may be closed by strictures, or may be obstructed by the formation of dacryoliths, by the growth of polypi, or by the entrance of foreign bodies; the lacrymal sac is subject to acute and chronic inflammation, which may eventuate in the formation of a lacrymal fistula; while the walls of the nasal duct are often the seat of inflammatory processes which tend strongly to occlusion of its lumen. New growths rarely originate in the drainage apparatus, but not infrequently involve it through extension from neighboring parts.

These various abnormalities of the different parts of the drainage apparatus are characterized by a common symptom: the passage of the tears from the conjunctival sac to the nose is interfered with, and in consequence they flow over the lid-margin and down upon the cheek. This condition, known as epiphora, or stillicidium lacrymarum, not only gives rise to much inconvenience *per se*, but not infrequently sets up a chronic conjunctivitis, which may be attended by eczema of the lids and cheek.

It is an interesting observation that women suffer much more frequently than men from affections of the lacrymal apparatus, the proportion being, according to Power, "about two or even three to one." Power gives the following statistics bearing upon this point: Nieden, he says, found in his practice the cases of lacrymal disease divided thus, 62.1 per cent. females and 37.9 per cent. males; Von Hasner found 74.5 per cent. females, 25.5 per cent. males; Schirmer, 67 per cent. females, 33 per cent. males. The explanation of this greater predisposition of women to lacrymal disease, he thinks, may be found in their sedentary occupations, their greater proclivity to tears, and perhaps in the smaller calibre of all the ducts, which, he believes, more than counterbalance the greater exposure to variations of weather to which men are subjected.[3] On this point Fuchs naïvely remarks[4] that the difference in the susceptibility to lacrymal affections of the two sexes is probably to be accounted for by

[1] Wien. Med. Presse, 1878, Bd. xix. p. 110.

[2] Bull. et Mém. Soc. de Chir. de Paris, 1876, N. S., vol. ii. p. 592.

[3] Lectures upon Diseases of the Lacrymal Apparatus, London Lancet, 1886, vol. ii.

[4] Text-Book of Ophthalmology, p. 520.

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the "zealous use which the female makes of the lachrymal apparatus." Nieden has advanced evidence to show that heredity is an important factor in the causation of lacrymal disease, which is in accordance with my own experience. He asserts that it exists in nine per cent. of all lacrymal affections, and that the transmission occurs much more frequently through the mother than through the father, the proportion being 63.3 per cent. from mother to child and only 36.7 per cent. from father to child. (Power.)

Atresia of the Lacrymal Puncta. — This condition occurs both as a congenital and as an acquired anomaly. Only a few cases of congenital atresia of the puncta have been reported, and in some of these the evidence as to the prenatal origin of the defect does not seem to be conclusive. In some of the reported congenital cases there was obliteration of the corresponding canaliculus as well as of the punctum, but in others the atresia was limited to the punctum. In none of them does it appear that the atresia involved all the four puncta. In an interesting case, occurring in a lad ten years of age, reported by Dr. S. M. Burnett,[1] both puncta of one eye and the lower punctum of the opposite eye were occluded. There was epiphora only in the eye in which both puncta were involved. An incision showed that the canaliculus was present in the lower lid of this eye, and it was slit up with relief to the epiphora. The evidence in favor of the congenital origin of this case seems to be fairly conclusive. Other cases have been reported by Zehender[2] and by Hugo Magnus.[3]

A case of complete and probably congenital atresia of the lower punctum of one eye, with epiphora of "years' duration," recently occurred in my own practice (April, 1894). So far as could be determined, the corresponding canaliculus also was absent. There was not even the usually observed depression to mark the site where the punctum should have been. All efforts to find an opening by means of small probes failed, and a sharp-pointed knife was introduced at the point where it seemed that the punctum ought to have been situated, and was carried on towards the lacrymal sac, as in the usual operation of slitting the canaliculus. Through the incision thus made an unsuccessful effort was made to pass a probe into the lacrymal sac. The sac was then entered with a sharp-pointed knife, and after this a No. 5 probe was passed into the sac and through the nasal duct, revealing the presence of complete stenosis of the duct, the strictures being of bony consistence. No especial difficulty was experienced after this in probing the duct through the passage thus made, although later the artificial canaliculus disappeared and the probes were passed directly into the lacrymal sac. In time a No. 16 probe (four millimetres' diameter) was introduced. A complete and doubtless permanent cure seems to have been effected in this case, as an interval of eight weeks was allowed to elapse between the

[1] Archives of Ophthalmology, vol. xiii. p. 53.

[2] Zehender's Monatsbl. f. Augenheilk., Bd. v. p. 131; and also in the number for January, 1884.

[3] Ibid., Bd. xiii. p. 176; also in Hirschberg's Centralbl. for April, 1880.

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last two probings without any difficulty being experienced in passing the probe which had previously been introduced, and twenty-one months after this last probing the duct was freely pervious to air and there was an entire absence of epiphora. The patient was a young woman, a Hebrew, about twenty years of age. The lower punctum of the opposite eye, it may be remarked, was exceptionally small.

Complete obliteration, or atresia, of the puncta, as an acquired condition, seldom occurs except as the result of traumatism, such as laceration of the lid or its partial destruction by lime or other caustic agent. Not infrequently, however, in ectropion and also in simple eversion of the punctum, particularly when associated with stricture of the canaliculus or lacrymal duct, the lower punctum, especially, becomes more or less completely occluded by an overgrowth of epidermis and by a gluing together of its walls. The puncta are also said to become occluded as a result of blepharitis marginalis and chronic conjunctivitis; but I do not remember to have met with such cases, except when ectropion or eversion of the puncta was also present, the desiccation of the tear-points which occurs when either of these two conditions exists seeming to be an important factor in bringing about the occlusion. Complete obliteration of the punctum has been known to occur as a result of the cicatrization of a small-pox pustule, and also has been met with as a consequence of chancre of the eyelid. (Larebière.)

FIG. 3.

Whether atresia of the punctum be congenital or acquired, it is not usually a difficult matter to remedy, provided the corresponding canaliculus is patulous. The most difficult cases to benefit are those of traumatic origin in which the injury has involved the canaliculus as well as the punctum, and the congenital cases in which the canaliculus is absent. Generally a slight depression marks the site of the occluded punctum, and, taking this as a guide, it is usually possible, by means of a straight, moderately sharp-pointed probe (Fig. 3), to drill an opening into the canaliculus, through which larger probes can subsequently be introduced. When the occlusion is attended by eversion of the punctum the canaliculus should be slit, as simple dilatation by means of probes is likely to prove of but little benefit; and, indeed, in most cases of atresia of the punctum this simple procedure is the one which affords the best and most permanent results. Another method of dealing with these cases, when only one punctum is occluded, suggested by Streatfeild, is to slit the canaliculus belonging to the unoccluded punctum, and to pass a small, properly bent probe through this passage into the lacrymal sac, and thence into and along the canaliculus of the occluded punctum. If practicable, the point of the probe is to be forced through the obstruction; otherwise it is to be cut down upon. After this the canaliculus may be slit in the usual manner.

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Besides atresia, there are other congenital anomalies of the puncta which are occasionally encountered. *Double puncta* and *double canaliculi* have been met with, and in connection with absence of the puncta the canaliculi have been represented by simple furrows along the edge of the lid. Von Graefe, F. Raab, Emmert,[1] and others have reported cases of double puncta.

There are several varieties of *malposition of the puncta*, which are usually observed in the lower lid, and which, by preventing the entrance of the tears into the canaliculi, give rise to epiphora. The puncta may be partially or completely everted, so that they are no longer in contact with the eyeball; they may be so decidedly inverted that a similar effect is produced; or, owing to the small size or deeply set position of the eyeball, they may be not in apposition with it, a triangular space intervening between the inner extremity of the lids and the surface of the eyeball. It is probable that the lower puncta perform a more important part in draining the tears from the conjunctival sac than the upper ones, and hence it is that their occlusion or malposition is of especial moment.

Eversion of the puncta is, of course, present in most cases of ectropion. It may occur also as a result of blepharitis, especially if this be attended by thickening of the margin of the lid. It is often met with in old age, in consequence of relaxation of the tissues of the eyelid and of loss of tone in the orbicularis palpebrarum, and it is a common accompaniment of facial paralysis. It is not infrequently produced by contraction of the external integument of the lid in the neighborhood of the puncta, such as may result from a slight traumatism or an eczematous or other inflammation. The malposition of the puncta is usually aggravated by the epiphora to which it gives rise; for the constant overflow of tears excites an inflammation in the skin of the lid and cheek, which is followed by a contraction such as has just been described.

Inversion of the puncta, as a condition requiring especial attention, is seldom met with. It is present in most cases of entropion, and, like entropion, is usually consequent upon chronic trachoma.

The efficient remedy in all malpositions of the puncta is the slitting of the canaliculus. This applies more especially to the lower canaliculus; for, judging from my own experience, the slitting of the upper canaliculus is very rarely called for. When the eversion of the lower punctum is so marked that the simple slitting of the canaliculus does not accomplish all that is desired, we may increase the effect of the operation by excising with slender curved scissors the inner lip of the divided canaliculus and a small bit of the adjoining conjunctiva, as suggested by Critchett. Usually the edges of a divided canaliculus show but little disposition to grow together, and if separated once or twice with a greased probe, at intervals of twenty-four or forty-eight hours, cicatrize and remain apart. I have, however,

[1] Archives of Ophthal. and Otol., vol. v. p. 525.

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occasionally met with instances in which they showed a most persistent tendency to reunite, in spite of repeated efforts to keep them apart. Under such circumstances it may become necessary to snip off the inner lip of the canaliculus, as just described.

Conjunctivitis, more or less marked, and affecting especially the palpebral conjunctiva of the lower lid, is apt to be present in most cases of eversion of the puncta; and while, as a rule, the simple slitting of the canaliculus will in time cause it to disappear, it is better to hasten its cure by prescribing a mild astringent collyrium. The one which I have found most useful consists of half a grain of sulphate of zinc and ten grains of boracic acid to an ounce of distilled water. If blepharitis be present, the ointment of the yellow oxide of mercury (gr. ii to dr. i) should be prescribed; and when this fails to act well, which it seldom does, an ointment of aristol of similar strength will often be found efficacious.

Atresia of the canaliculi, as has already been mentioned, may occur as a congenital defect in association with occlusion of the puncta, and also as a consequence of traumatism involving the region of the inner canthus. Circumscribed strictures of the canaliculi are met with not infrequently, especially in connection with stenosis of the lacrymal duct. They may be located at any part of the canal, but are usually found near its inner extremity, at the point where it enters the lacrymal sac, or where the upper and lower canaliculi join just before entering the sac. When there is complete obliteration of the canaliculus, its restoration by operative procedure is impracticable. It may be possible, however, by repeated probings, to open a passage-way for the tears directly into the lacrymal sac, as was accomplished in the case above related. When the occlusion is circumscribed, it may usually be overcome by means of a small probe: in doing this, however, some caution is necessary, lest a false passage be made. The lid should be put upon the stretch, and the probe (No. 2 being the size best adapted for this purpose) should be introduced into the puncta and passed along the canaliculus towards the sac in the usual manner. Should the force which it is possible to exert with such a probe fail to overcome the stricture, we may succeed better with a straight, moderately sharp-pointed probe (Fig. 3), to which it is practicable to give a rotary motion. If this too should fail, we must cut through the stricture and enter the sac with a sharp-pointed knife. The old-fashioned, Sichel cataract-knife I have found especially well adapted for this purpose. The use of this knife necessarily involves the slitting of the canaliculus, but this is not an objection; for, even when the stricture has been overcome by means of a probe, it is best, as a rule, afterwards to divide the canaliculus with the probe-pointed knife, this being the most effectual way of preventing a recurrence of the occlusion.

The so-called *dacryoliths*, which occasionally form in and obstruct the canaliculi, were formerly supposed to be simply concretions of lime. They are now known to be composed in great part of a fungus, believed by some

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investigators to be identical with the leptothrix buccalis, which inhabits the cavity of the mouth. Cohn, however, to whom Foerster submitted one of these concretions for examination, denies that the two are identical, and suggests the name "streptothrix Foersteri" for the lacrymal fungus. Goldzieher has met with cases in which a cilium occupied the centre of the dacryolith, and he believes that their development is often due to the lodgement of an eyelash in the canaliculus.[1] They vary in color, being sometimes of a yellowish or greenish white, and again gray or even grayish black; they may be several millimetres in length and one and a half millimetres or more in diameter; occasionally they project through the punctum. Foerster has reported a case in which a persistent conjunctival inflammation was kept up for years by the presence in the lower canaliculus of one of these concretions.[2] An analysis of a dacryolith made by Mr. Curtis showed that it consisted of 28.3 parts of organic matter and 71.7 parts of calcium phosphate. (Power.) The presence of a dacryolith in the canaliculus may be detected by the slight circumscribed swelling to which it gives rise. It should, of course, be removed as soon as detected, and to effect this the canaliculus will usually require to be slit.[3]

Polypi have been met with (but very rarely) in the canaliculi. They produce epiphora, and in consequence of this may set up a chronic conjunctivitis. Their removal may necessitate the division of the canaliculus.

Small *foreign bodies* occasionally find their way into the canaliculi, where they may remain for a considerable time. Eyelashes not infrequently enter the punctum, and sometimes pass wholly into the canaliculus; at other times a portion of the lash projects from the punctum and, coming in contact with the eyeball, produces decided discomfort, as I have had occasion to observe more than once. Cases have been reported in which bits of the beard of barley and of wheat have been found lodged in the canaliculus, and one case (by Haffner) in which an ascaris lumbricoides was removed from the lower punctum.[4]

Dacryocystitis, or *inflammation of the lacrymal sac*, occurs, as has already been said, as a chronic and also as an acute affection. Primary inflammation of the lacrymal sac is extremely rare. In the vast majority of cases the inflammation is secondary to and dependent upon disease of the nasal duct, stricture of the duct being the condition which usually gives rise to it. Primary acute dacryocystitis is said to occur occasionally in strumous children, and it may also be produced by external violence or by the entrance into the sac of an irritant fluid. Cases of this character are, however, very rarely encountered.

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[1] Centralbl. f. Prakt. Augenheilk., February, 1884.

[2] Archiv f. Ophthal., Bd. i. p. 284.

[3] Keyser, of Philadelphia, has reported three cases of "leptothrix" in the upper canaliculus (Med. and Surg. Reporter, Philadelphia, vol. xliii. p. 28, 1880), and Gruening one case of like character (Arch. of Ophth. and Otol., New York, vol. iii. p. 17, 1873).

[4] Berlin. Klin. Wochensch., 1880, Bd. xvii. p. 346.

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Fig. 4.

Acute dacryocystitis.

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Inflammation of the lacrymal sac, secondary to disease of the nasal duct, usually begins not as an acute but as a mild chronic affection, the so-called blennorrhœa or catarrh of the sac, which is unattended by pain, and makes itself manifest chiefly through the accumulation of tears and mucus in the sac, their regurgitation through the puncta, and the existence of epiphora. Acute exacerbations of the catarrhal inflammation, characterized by the formation of pus, are liable to occur from time to time, and constitute what is known as abscess of the lacrymal sac, or acute dacryocystitis. It is possible, of course, for this sequence of events to be reversed, and for an inflammation extending from the nasal duct into the sac to manifest itself first in the form of an acute attack, but my experience leads me to believe that this is less frequently the case.

The history of most cases of dacryocystitis is as follows: Inflammation of the walls of the lacrymal duct (usually secondary to nasal disease), leading to more or less complete occlusion of the duct; in consequence of this, accumulation of tears in the duct above the point of occlusion and in the lacrymal sac; the retained tears, owing to the entrance of bacteria from the conjunctival sac, presently undergoing putrefactive changes, and, as a result of this, irritating the mucous lining of the sac and duct and exciting in it a catarrhal inflammation; the mucus collecting in the sac, as a consequence of this, forming a medium better suited to the growth of the intruding bacteria, and thus supplying the conditions most favorable to the development of a chronic and persistent inflammation. In some instances this state lasts indefinitely without undergoing appreciable change; but in others, whether through the occurrence of a slight traumatism, exposure to cold, the entrance into the lacrymal sac of micro-organisms of unusual virulence, some constitutional disturbance, or, as seems to happen occasionally, the sudden occlusion of the canaliculi at their point of juncture with the sac, the inflammation undergoes a rapid change in character and assumes an acute form. (Fig. 4.) Severe pain, accompanied by tense swelling of the sac and marked œdema and redness of the lids and surrounding parts, comes on; thick, creamy pus forms in the sac, and not infrequently decided evidences of constitutional disturbance, such as fever, preceded perhaps by rigor, loss of appetite for food, and sleeplessness, manifest themselves. After several days of intense suffering the integument over the sac assumes a yellowish appearance, becomes thin and bulging, and, if left to itself, usually gives way at a point about corresponding to the lower margin of the orbit (beneath the internal palpebral ligament), permitting the contents of the sac to escape and affording the patient almost immediate relief from suffering. In exceptional instances the inflammation subsides without perforation of the sac occurring, the pus ultimately escaping through the canaliculi and puncta. In other exceptional instances the perforation occurs through the inner wall of the sac: under such circumstances the pus has been known to find its way into the nasal meatus and even into the maxillary antrum, and in one case,

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reported by Power,[1] it burrowed "between the periosteum and the bone, till at length it reached the floor of the nose and established a fistulous orifice into the mouth through the palatine suture of the palatal bones." Other cases have been observed in which the pus, after escaping from the sac, has burrowed beneath the integument of the face and has finally perforated the skin near the ala of the nose or at some distant point upon the cheek. (Velpeau.)

As the inflammation of the sac declines and the discharge diminishes, the opening through which the pus has escaped, whether it has occurred spontaneously or has been made artificially, gradually lessens in size, and in the course of a week or two usually closes. This fortunate result does not, however, always happen; for in some cases the continual discharge of tears through the opening prevents its healing, and thus a permanent fistula—a second variety of fistula lacrymalis—is formed, and becomes a source of much annoyance to the patient. With the subsidence of the acute inflammation, whether a fistula remains or not, the sac returns to its previous condition of chronic catarrh.

In chronic dacryocystitis there is, as has already been mentioned, an almost constant regurgitation of muco-purulent matter through the puncta into the conjunctival sac, and, as a result of this, conjunctivitis and keratitis are often met with as secondary complications. Blepharitis marginalis and eczema of the lower lid and cheek are also frequently produced by the overflow of tears and muco-pus, and ultimately partial ectropion of the lower lid may ensue from the contraction of the external integument of the lid caused by the continued eczema. When a fistula has become established, although at first the pus may have found its way by the most direct route from the sac to the surface, the discharge is liable to burrow beneath the skin, and in this way, in time, quite extensive sinuses may be formed, with perhaps several fistulous orifices. Under such circumstances it is not uncommon to find more or less extensive caries of the underlying bone. These unpleasant complications do not exist in every case of chronic dacryocystitis; but, even without them, this affection gives rise to much discomfort because of the constant epiphora which attends it and the blurring of sight which the muco-purulent fluid flowing over the cornea produces.

Dacryocystitis being dependent in almost all cases, as has already been said, upon stenosis of the lacrymal duct, it follows that its treatment is for the most part the treatment of stricture of the duct, of which we shall speak presently, after we have considered the pathology of this latter affection. Something may be said here, however, as to the treatment of the acute form of dacryocystitis. If a case of acute inflammation of the lacrymal sac is seen in its incipiency, an effort may be made to cut short the attack. An energetic calomel cathartic should be given, and this may

[1] In his lectures upon Diseases of the Lachrymal Apparatus, London Lancet, 1886, vol. ii, p. 855.

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be followed up by liberal doses of quinine or of pyrophosphate of sodium (gr. xv to xx every two or three hours). The local treatment should consist of the application on a linen or gauze pad of a lotion of opium and boracic acid (ext. opii, gr. x to xv ; acid. boracic., dr. i ; aquæ destill., oz. iv), or one of opium and acetate of lead (ext. opii, gr. x to xv ; plumb. acetat., gr. xv to xx ; aquæ destill., oz. iv), and, perhaps, the abstraction of blood by leeching. An anodyne for the relief of the severe pain may also be required. If these measures fail to subdue the inflammation, warm poultices should be applied. The best are made of flaxseed-meal, to which a small quantity of laudanum and powdered boracic acid may be added with advantage. As soon as it is evident that pus has formed in the sac and is endeavoring to find a means of escape, an incision should be made directly into the sac through the thinned overlying integument. Such an incision, if made in the direction in which the skin tends to wrinkle,—that is, from above and toward the nose downward and outward,—leaves no perceptible scar, and gives a more ready exit to the pus than does an incision made into the sac along the canaliculus. As the inflammation and discharge subside, the poulticing should be discontinued and one of the lotions just mentioned prescribed instead. A collyrium of bichloride of mercury (1 to 12,000) may also be ordered. No attempt should be made, however, until some days after the complete subsidence of the acute attack, to overcome the stenosis of the lacrymal duct by the passage of probes, as this, if done prematurely, is apt to induce a recurrence of the acute inflammation.

Stricture of the Lacrymal Duct.—In speaking of the anatomy of the lacrymal duct, mention has been made of the fact that its membranous walls partake of the character of both a mucous and a periosteal membrane, that they contain a dense plexus of veins resembling those of the turbinated bodies, and that at certain points they are thrown into valve-like folds which encroach considerably upon the lumen of the canal. Such being the case, it is evident that even a slight amount of inflammation of the walls of the duct must almost inevitably be attended by at least a transient occlusion of its lumen. If, moreover, we bear in mind that the lacrymal duct is in a sense but a part of the nasal cavity into which it opens, and that it holds as close a relationship to this cavity pathologically as it does anatomically, we can readily understand, since inflammatory affections of the nasal passages, both acute and chronic, are of such very common occurrence, why it is that stricture of the lacrymal duct is by no means a rare affection. While a certain number of cases of lacrymal stricture are of traumatic origin, the inflammation, under such circumstances, not infrequently beginning in the sac and extending thence to the duct, the large majority are, without doubt, secondary to and dependent upon acute or chronic nasal catarrh. Watering of the eyes is a well-known symptom of acute rhinitis, and probably in the majority of the pronounced cases of this affection the catarrhal condition involves to a greater or less

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degree the mucous membrane which lines the walls of the lacrymal duct. In the vast majority of cases, with the subsidence of the rhinitis the catarrh and transient occlusion of the duct disappear and the parts return to their previous normal condition. In exceptional instances, however, because of the severity of the inflammation, the occurrence of a second or third attack before the first has been recovered from, a congenital narrowness of the duct, or a peculiar susceptibility of the lacrymal passages to disease (a susceptibility which in some instances is undoubtedly inherited), the inflammation of the duct does not subside with the nasal affection, but assumes a more serious and chronic character. Under such circumstances the nature of the inflammation soon changes, and what was at first simply a catarrh of the mucous membrane presently becomes an inflammation of the periosteum, and the temporary occlusion of the duct from engorgement of the submucous plexus of veins gives place to a permanent stenosis, due to plastic effusion and periosteal, and at a still later stage osteal, thickening.

While acute nasal catarrh gives rise, in the manner just described, to some cases of stricture of the lacrymal duct, probably a much larger number are dependent upon chronic inflammatory affections of the nasal passages. The chronic nasal affections of inherited and acquired syphilis are especially apt to involve the lacrymal apparatus. Syphilis has long been recognized as a cause of disease of the lacrymal sac and duct, and doubtless this happens in most instances through the extension of specific inflammation from the nose.

Seventeen out of two hundred and forty cases of stricture of the lacrymal canal, noted in Galezowski's clinic, were found to be of syphilitic origin. (Larebière.[1]) The same author also points out that syphilitic gummata occasionally develop in both the lacrymal sac and duct. Power remarks that "lachrymal obstructions are not rare in infants presenting the usual manifestations of secondary syphilitic disease of the mucous membrane of the mouth and fauces." In the same connection he states that the mucous membrane of the lacrymal sac, in many instances, "becomes affected by the extension of disease [syphilitic] of both the conjunctival and nasal mucous membrane."[2] Upon the point of the extension of inflammation from the conjunctiva to the lacrymal sac I am disposed to take issue with him; for, while my experience leads me to believe that there is the closest pathological sympathy between the lacrymal sac and the nasal mucous membrane, it has convinced me that quite the reverse is true as to the lacrymal sac and the conjunctiva. In support of this view no better evidence can be adduced than that in so virulent a disease as gonorrhœal ophthalmia the lacrymal passages never become involved in the inflammatory process. Tuberculosis of the nasal mucous membrane, through extension to the lacrymal passages, is an occasional cause of stricture of the

[1] Thèse de Paris, 1880.

[2] Lectures upon Diseases of the Lachrymal Apparatus, already cited.

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duct. Polypi have been met with in the lacrymal sac, and by obstructing the passage of tears into the nose they may give rise to symptoms resembling those of stenosis of the duct. They may also, in time, through the irritation which they produce and the damming up of the tears, promote the development of actual stricture.

Power thinks that stricture of the lacrymal duct is not infrequently dependent upon periostitis of the alveolar process due to carious teeth, especially the canines. The suggestion is worth bearing in mind, though I am not inclined to accept the view that lacrymal stricture is often produced in this way.

When once established, stricture of the lacrymal duct due to periosteal or osteal thickening never disappears spontaneously, but, with all its unpleasant consequences,—epiphora, blennorrhœa of the sac, recurrent attacks of acute dacryocystitis, secondary inflammation of the conjunctiva and cornea, and perhaps lacrymal fistula,—lasts for a lifetime. Such being the case, it is not surprising that the treatment of this affection, even from the time of the ancients, should have received the serious consideration of physicians and surgeons. A volume would be required to give in detail an account of the various operative procedures which have been proposed from time to time for the cure of stricture of the lacrymal duct, or for the relief of the consequences to which it gives rise. Many distinguished surgeons have considered this affection worthy of their study, and among those who have suggested methods of treatment or have modified those previously in vogue may be mentioned Anel, Petit, Wathen, Woolhouse, Ware, Scarpa, Dupuytren, Beer, Desault, Travers, Desmarres, Hays, Bowman, Critchett, Teale, Weber, Stilling, Noyes, H. W. Williams, Green, E. Williams, and Couper.

The various surgical procedures which have been practised may be classed under four heads: 1. Those which aim to restore the natural passages. 2. Those which have for their object the formation of a new passage into the nose for the tears. 3. Those which aim at the obliteration of the natural passages,—the lacrymal sac and duct. 4. The removal of the lacrymal gland for the purpose of arresting the secretion of tears.

Probably the oldest of these different procedures is that which aims at the obliteration of the natural passages. More than eighteen hundred years ago, in Rome, as Celsus tells us, the practice of destroying the lacrymal sac and duct by means of the actual cautery was in vogue. Seven hundred years later this same procedure was revived by Paulus Ægineta, and again it was revived in 1748 by Nanoni at Florence, whence it spread to Germany and France. In more recent times the obliteration of the lacrymal sac has been effected by the use of caustic agents, such as nitrate of silver, chloride of zinc, nitric acid, Vienna paste (as employed by Desmarres), caustic potash, etc., and still more recently by the thermo-cautery, by the galvano-cautery, and by excision. The merit claimed for this procedure is that it relieves the patient from inflammation of the

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lacrymal sac and its unpleasant consequences, and that in some instances it even cures the epiphora through the inhibitory influence, already referred to, which it appears to exert upon the activity of the lacrymal gland. Desmarres, though he employed this method and speaks favorably of the results which he obtained from it in certain intractable cases, declares that it should be regarded as "une dernière ressource," and adds that if a cure can be effected by other means, sound practice counsels its employment,[1]—an opinion with which I am heartily in accord.

The usual method of performing this operation at the present day is to make a free incision into the lacrymal sac through the external integument and the internal palpebral ligament, and through this to introduce the caustic agent or the tip of the galvano- or thermo-cautery, a Manfredi's speculum being employed, if either form of cautery is used, to protect the lips of the wound. Excision of the lacrymal sac, owing to the delicate structure of the sac-walls, is not an operation easy of performance. A vertical incision of sufficient length is made through the skin down to the sac, and the latter is dissected out as carefully and completely as possible with a scalpel or a pair of blunt-pointed scissors. After this the cavity left by the removal of the sac and the upper part of the duct should be scraped with a sharp spoon, and then, the wound having been cleansed with an antiseptic solution, the edges of the external incision should be closed accurately with stitches and a suitable aseptic dressing applied.

The making of an artificial passage directly from the lacrymal sac into the nose, by perforating the os unguis, is an operation which also dates back to classical times. In more recent times it was practised, and the procedure variously modified, by Woolhouse, Hunter, Laugier, and Wathen. In the operation as performed by Woolhouse a gold canula was introduced into the opening through the os unguis, in order to prevent the closure of the artificial passage. The procedure is one which has for us only an historical interest, as it is never practised at the present day.

Much ingenuity has been displayed, and by many eminent surgeons, in devising means to restore the natural lacrymal passages when they have become occluded by accident or disease. More than a century ago (1781) the plan of introducing into the strictured lacrymal duct a canula, made of gold or silver, was suggested by Wathen in England, and until quite recent times this procedure, which was revived by Dupuytren, was extensively practised. The canula, which was to be worn permanently in the duct and afford a passage-way for the tears, was introduced through an incision made directly into the lacrymal sac below the internal palpebral ligament. Its upper extremity was made flange-shape, in order to prevent its falling through the duct into the nose. In spite of this precaution, however, it usually fell out sooner or later, generally finding its way into the nasal cavity, or, if this did not happen, it became obstructed by cal-

[1] Maladies des Yeux, Paris, 1847, p. 891.

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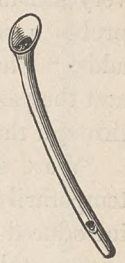
careous matter, so that its usefulness was in a great measure destroyed. So long as it remained in the duct it afforded a measure of relief, though it did not always do away with the epiphora or the blennorrhœa of the sac. Its curative effect was slight, and when, after having been worn for some time, it fell out, as has been said it usually did, the duct soon became re-occluded, and the condition of the patient was presently as bad as before the introduction of the canula.

This method of dealing with lacrymal obstructions was the one usually employed by my grandfather, Professor Nathan R. Smith, and the accompanying illustration (Fig. 5) represents a gold canula, now in my possession, which he placed in the lacrymal duct of a female patient in 1858, and which remained in position for twenty-five years. It was still being worn when I first saw this patient, in August, 1883; but, a short time afterwards, while she was engaged in hanging pictures, it fell into the nose and then into the pharynx, and was removed through the mouth. Previous to its introduction the patient had suffered from recurrent attacks of acute dacryocystitis, which she was free from during the twenty-five years that the canula was worn. She was still annoyed during this period, however, by epiphora and by chronic catarrhal inflammation of the sac. The lower extremity of the canula is filled with calcareous material which completely obstructs one of the two openings near its tip and partially obstructs the other, as is shown in the illustration. Its walls also are worn through in several places. This patient was afterwards treated by me with large probes, and a complete and permanent cure was effected. Another case has come under my observation in which a gold canula, after having been worn for some time, passed through the floor of the nose and into the alveolar process, and was finally removed through the socket of one of the incisor teeth, which had been previously extracted. At the present day this method of dealing with occluded lacrymal canals is, so far as I am aware, never resorted to.

FIG. 5.



Gold canula worn for twenty-five years.

According to Desmarres, the credit of having first suggested for the cure of lacrymal stricture the use of a contrivance intended to be worn *temporarily* in the lacrymal duct is due to J. L. Petit. His plan was to make an incision into the lacrymal sac, pass a grooved director through the duct, and, by the aid of the director, to introduce a bougie, which was changed daily. Desmarres practised essentially this same method. Anel attempted to overcome the obstruction of the duct by forcing water through the canaliculus into the sac by means of the syringe which bears his name. He also endeavored to dilate the strictures by means of slender probes which he introduced through the canaliculus; but he regarded this method as serviceable only in cases of slight obstruction. Benjamin Travers, who was very sceptical as to the utility of the gold canula, which Dupuytren used so extensively during his time, also employed probes, which

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he passed, as Anel did, through the punctum and canaliculus. His probes were larger, however, than those of Anel, and his results were, therefore, more satisfactory. He also employed slender styles with "flat heads, gently sloped," which he introduced through the canaliculus, permitting them to remain in position only twenty-four hours because of their tendency to cause ulceration of the punctum. The late Dr. Isaac Hays, of Philadelphia, early adopted the plan of introducing probes through the canaliculus, and he modified and improved the probes of Travers. He used probes which varied in size from the thickness of No. 21 to that of No. 17 wire (English standard gauge), and contended (in a letter which I received from him several years before his death) that he had obtained very satisfactory results. He writes, "I have cured *permanently* with my probes a very large number of cases of lacrymal obstruction." And he adds, "I have found no difficulty in gradually dilating the lower punctum and the passage to the sac; the difficulty always was in passing the probe through the ductus ad nasum."

Ware suggested the use of nail-headed styles, which were to be worn temporarily, with the expectation of curing the stricture. They were introduced through an incision in the lacrymal sac, the round, flat head of the style being permitted to remain outside the opening. Beer employed catgut cords of different sizes, which he introduced into the duct in a similar way and passed slowly through it, a fresh portion of the cord (which was kept coiled upon the head) being drawn into the duct each day, while the part which had previously been in the duct was drawn out through the nose and cut off. Méjean used meshes of silk threads, which he passed through the canaliculus and duct by means of a slender, needle-like probe. Blizzard practised a still more novel plan. He filled the lacrymal sac with quicksilver, and expected the weight of the small globule of mercury which the sac is capable of holding to overcome the stenosis of the duct. Probes intended to be passed from the nose through the inferior orifice of the lacrymal duct were also contrived, and great advantages claimed for them; but they were not received with favor.

The operation, devised by Bowman, of slitting the canaliculus, to facilitate the introduction and passage of lacrymal probes, constitutes a distinct advance over all previous methods of dealing with strictures of the nasal duct, and may be said to mark the beginning of a new era in the surgery of the lacrymal apparatus. There are several methods of performing this simple operation. Bowman passed a slender grooved director, which he devised for the purpose, through the canaliculus, and, using this as a guide, slit the canaliculus with a cataract-knife or other suitably shaped, sharp-pointed knife. He also contrived a narrow, probe-pointed knife with which he divided the canaliculus without making use of the director. Weber devised a beak-pointed canaliculus-knife (Fig. 6) which was received with more general favor, and which, in its original or slightly modified form (Fig. 7), is commonly employed at the present day. Slender

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scissors having one blade longer than the other have also been used to divide the canaliculus. My own preference is for the straight, probe-pointed knife, shown (though with somewhat too broad a blade) in Fig. 7; and I invariably divide the *lower* canaliculus when the purpose of the operation is to facilitate the passage of probes through the lacrymal duct.

FIG. 6.

Weber's canaliculus-knife.

FIG. 7.

Probe-pointed canaliculus-knife.

To divide the canaliculus properly some little skill and experience are required. It is especially important that the edge of the knife should not be inclined forward, otherwise a slight, perceptible deformity will result, and, furthermore, the position of the divided canaliculus will not be the most favorable for the carrying off of the tears. The operator should stand behind the patient, letting the patient's head (covered with a napkin) rest against his chest, the left hand being used for the left eye and the right hand for the right eye, the lid being kept tightly upon the stretch with the thumb of the opposite hand. The probed tip of the canaliculus-knife should be introduced vertically into the punctum (which, together with the canaliculus, should have been dilated previously by the passage of one or two of the smallest-sized probes), and then, the direction of the knife having been changed, it should be passed horizontally along the canaliculus and into the lacrymal sac until its progress is arrested by the inner wall of the sac supported by the lacrymal bone beyond. This point having been reached, and the edge of the knife being directed upward, or upward and slightly backward, the lid being kept still tightly on the stretch, the canaliculus is divided by simply elevating the handle of the knife. If the operation is done as a preparatory step to the treatment of stenosis of the lacrymal duct, the canaliculus should be divided well up to its juncture with the sac; but if done for some other purpose, as, for example, eversion of the punctum or stricture of the canaliculus, it is not necessary to carry the division quite to this point.

In most cases the edges of the divided canaliculus have a tendency to unite, and occasionally some difficulty is experienced in overcoming this tendency. Usually, if they are separated once or twice by the passage of a greased probe, at intervals of forty-eight hours, reunion will be permanently prevented. I have seen cases, however, in which it was necessary to repeat the probing a number of times before the desired result was obtained; while, on the other hand, it is not uncommon to find that the cut edges, from the first, show no disposition whatever to grow together. A few

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instillations of a four per cent. solution of cocaine render the operation of division of the canaliculus almost painless.

Although the great value of Bowman's operation of slitting the canaliculus, when employed as a preliminary measure to the treatment of stenosis of the lacrymal duct, consists in the fact that it permits much larger probes to be passed through the duct than can be introduced through the undivided canaliculus,—probes, indeed, sufficiently large to restore completely the normal calibre of the duct,—Bowman himself fell far short of fully appreciating this, as is shown by the fact that the largest of the series of lacrymal probes which he employed (No. 6) had a diameter of scarcely one and a half millimetres, not quite as large as the biggest of the probes (corresponding in size with No. 17 of the English standard wire gauge) which Dr. Isaac Hays had been in the habit, previously, of introducing through the undivided canaliculus.[1] Such being the case, it is not surprising that his results in dealing with strictures of the lacrymal duct, and the results of those who followed strictly his plan of treatment, should have been far from satisfactory. A distinct advance over older methods had undoubtedly been made; but, after all, it was but one step—a long one, it is true—in the right direction. With probes of such small size as he employed the strictures necessarily were very imperfectly dilated, and as a consequence of this relapses occurred, after the discontinuance of the treatment, with discouraging frequency.

As a result of this, various modifications of Bowman's method were proposed. Pridgin Teale, of Leeds, Critchett, and Dr. H. W. Williams, of Boston, employed probes of about the size of Bowman's, but with bulbous extremities, while Couper, of London, used bougies of laminaria digitata. Stilling made a more radical departure by recommending free incision of the strictures[2] by means of a knife which he contrived for the purpose, and which he passed into the duct through the divided canaliculus. Warlomont followed Stilling's example, and reported good results; but others were not so fortunate, and this method has never come into general favor. Dr. Herzenstein proposed the forcible dilatation of the strictures upon the principle of Holt's dilatation of urethral strictures.

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[1] Dr. John Green, in his paper upon the use of lead styles (Trans. Am. Oph. Soc., 1867–68, p. 34), gives the diameter of Bowman's No. 6 probe as 1.3 millimetres, and that of Dr. Hays's largest probe, corresponding to No. 17 wire, as 1.5 millimetres.

[2] It has recently come to my knowledge (since the above was written) that as early as 1846 my grandfather, Professor Nathan R. Smith, practised division of lacrymal strictures. Whether the operation was original with him I am unable to say; but, in a copy of "Lawrence on the Eye," presented to the library of the Johns Hopkins University by the late Professor Christopher Johnston, I found by accident an interleaved illustration, drawn in ink by Professor Johnston, representing a knife which my grandfather had contrived for the division of lacrymal strictures. The illustration, which is faithfully reproduced in the accompanying wood-cut (Fig. 8), bears this inscription: "Prof. N. R. Smith's knife for dividing a stricture of the lacrymal duct. 1st Nov. '46." Doubtless this knife was introduced, as probes were usually introduced at that time, through an incision made directly into the lacrymal sac.

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The use of styles of different patterns was also combined with the slitting of the canaliculus. Dr. E. Williams, of Cincinnati, reported favorable results from the use of silver styles of large size,[1] while Dr. Green, of St. Louis, recommended styles made of lead, because they could be easily fashioned to suit each case, and be made to adapt themselves to any irregularities in the shape or curvature of the duct.[2] Instead of the nail-head of the older form of style, all of these, as they were introduced through the divided canaliculus, had slender, curved necks, intended to be bent over the margin of the lid. According to Soelberg Wells, Bowman was the first to employ styles of this kind.[3]

FIG. 8.

Knife contrived by Dr. N. R. Smith for dividing strictures of the lacrymal duct.

Dr. E. Williams, of Cincinnati, was probably the first, and, after him, Dr. H. D. Noyes, of New York, to realize the importance of accomplishing a more thorough dilatation of lacrymal strictures than could be effected by means of the probes employed by Bowman. Dr. Williams, as a preliminary step to the introduction of the large silver styles which he used, employed probes having bulbous, olive-shaped extremities. The largest of the styles which he used had a diameter of 2.9 millimetres, and the bulbous portion of the largest of his probes a diameter of 3.25 millimetres, considerably more than twice that of Bowman's No. 6. Dr. Noyes, probably as early as 1870, made use of short, hard-rubber probes, which in their higher numbers had a maximum diameter of four millimetres.[4] The biconical sound of Weber, which at its largest part has a diameter of about 3.25 millimetres, was in use some years before the date just mentioned; but, owing to its conical shape, this sound is capable of dilating widely only a small portion (about the upper third) of the lacrymal duct.

A very brief experience in the treatment of diseases of the lacrymal apparatus convinced me of the inadequate size of the probes recommended by Bowman, and satisfied me that, if success in dealing with strictures of the lacrymal duct was to be obtained, much larger probes than his must be employed. Accordingly, I began in the early part of 1874, without knowledge of what Dr. Williams and Dr. Noyes had already done in this direction, to add larger probes to the sets which I had previously used.[5]

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[1] Archives of Ophthal. and Otol., vol. i. p. 40, 1869.

[2] Trans. Am. Ophthal. Soc., 1867–68, p. 31.

[3] Diseases of the Eye, p. 69, Henry C. Lea's Son & Co., Philadelphia, 1883.

[4] Personal communication.

[5] The probes which I had employed up to this time were obtained in Europe in 1871, one set from a prominent instrument maker in Vienna, the other from one in London. They were of but six sizes, the largest in each set measuring 1.50 millimetres in diameter, and were similar to the probes which I had seen in use at the chief ophthalmic clinics of the two cities named.

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Three numbers were added at this time,—No. 7, having a diameter of 1.75 millimetres; No. 8, 2 millimetres; and No. 9, 2.12 millimetres,—and during the next two years I had made three still larger sizes,—Nos. 10, 11, and 12, the latter having a diameter of 2.75 millimetres. The advantage of employing these larger probes was soon made evident in the better and more permanent results which were obtained, and this led me in 1876, in noticing the then recently published "Treatise on Diseases of the Eye" by Brudenell Carter, to speak as follows: "In considering the subject of diseases of the lacrymal apparatus, he (Brudenell Carter) speaks in a very discouraging way of his success, or rather want of success, in relieving strictures of the nasal duct and the inflammatory conditions dependent upon them by means of the modern method of slitting the canaliculus and probing. I am at a loss to account for such unsatisfactory results as he describes, except upon the supposition that he has failed to recognize the importance of using much larger probes than were originally recommended by Bowman; for this, I am persuaded, is the secret of success in treating this troublesome malady."[1]

It was in the year following this (1877) that I undertook the measurements of the lacrymal duct which have been already described in treating of the anatomy of the drainage apparatus. My object in making these measurements was to ascertain the usual size of the lacrymal duct in its normal condition, and to learn whether it was not practicable to use still larger probes than I had ventured to employ up to that time; for I felt convinced from my previous experience that if this could be done still better clinical results would follow. Furthermore, I was anxious to substantiate the opinion which I had previously expressed (and which has just been quoted) as to the inadequate size of Bowman's probes, and I had little doubt that the measurements would show the calibre of the duct to be so great that the absurdity of attempting to dilate it thoroughly by probes of the size of those used by Bowman would be made evident. That such was the result of my investigation, I think, scarcely admits of question. The accompanying illustration (Fig. 9) shows in a striking manner the great disproportion between the size of the normal lacrymal duct, as shown by my measurements, and that of the largest of Bowman's probes,—No. 6. I may add that in crediting Bowman's No. 6 probe with a diameter of 1.50 millimetres I have probably somewhat overstepped the mark, for Soelberg Wells,[2] who ought to be good authority upon such a point, speaks of its being "about one-twentieth of an inch in diameter," which would be only 1.27 millimetres.

In the same paper in which the results of these measurements of the lacrymal duct were published[3] I described the series of probes which I had then

[1] Notes on the Progress of Ophthalmology, Trans. of the Med. and Chir. Faculty of Maryland, Baltimore, 1876.

[2] Diseases of the Eye, p. 619.

[3] Trans. of the Med. and Chir. Faculty of Maryland, 1877, p. 154.

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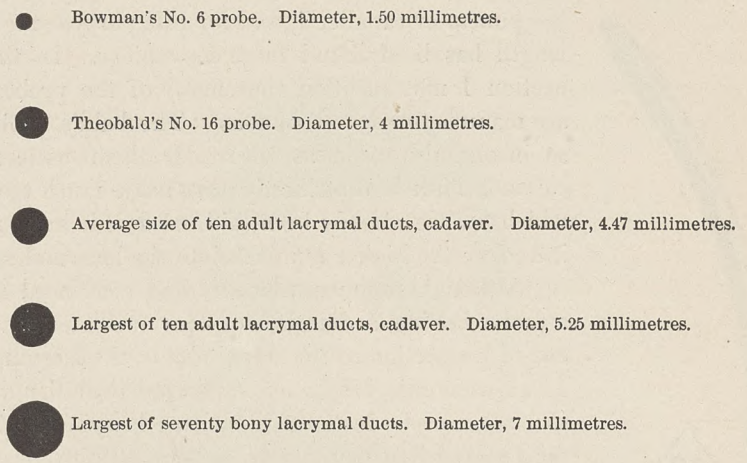
just begun to use, and which upon many occasions since I have commended to my professional brethren.[1] The especial features of these probes are the large size of the higher numbers, the shape of their ends, the number of probes composing the set, and the definite gradation of the sizes. The series comprises sixteen sizes. The smallest probe (No. 1) has a diameter

FIG. 9.



of .25 millimetre, the largest (No. 16) a diameter of 4 millimetres, the difference between the diameters of the successive numbers being .25 millimetre. The shape of their ends was regarded as a matter of importance; for it was evident that if they were made as square and blunt as those of the lacrymal probes in general use previously, the larger sizes would be useless, since it would be impossible to introduce them into the sac through the divided canaliculus. For this reason, and, moreover, to facilitate their passage through the strictured duct, the ends were made decidedly less blunt than those of Bowman's probes, being fashioned much like, but more pointed than, the smaller end of an olive, and with the same absence of shoulder. The proper shape of the ends, as well as the curve which has been found most convenient, is well shown in Fig. 10, which represents the actual size of the largest probes of the series (Nos. 15 and 16). The first sets that I had constructed were made of silver,—the larger sizes of pure silver, so that their shape might be more readily altered if occasion required, the smaller sizes of coin silver. Afterwards I had the larger sizes —from No. 7 to No. 16—made of aluminium,[2] because of the lightness and more slippery character of this metal. It is not tough enough to be used in making the smaller probes; but the larger probes made of it,

[1] Archives of Ophthal. and Otol., vol. vi.; Trans. Am. Ophthal. Soc., vol. ii., 1879; Reference Handbook Med. Sci. (Buck), vol. iv. p. 371; Trans. Eighth Internat. Ophthal. Congress, 1894.

[2] American Journal of Ophthalmology, vol. iv. p. 61, 1887.

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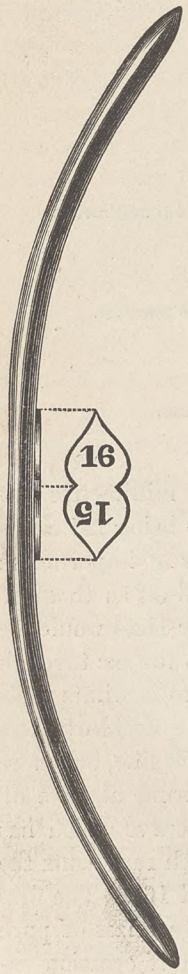
besides being lighter, meet with less resistance in being introduced and cause less pain than those made of silver. Nickel-plated copper probes I have also used with much satisfaction; they, like those made of aluminium, are more easily passed through the duct than the silver probes, but of course they have not the advantage of lightness. The No. 1 probe should be made about three-fourths of an inch shorter than the other sizes, as it is intended to be used only for dilating the puncta or canaliculi, and for this purpose the shorter length has been found more convenient. In this connection I may mention that many of the probes which are manufactured and sold as "Theobald's probes" are so incorrectly made as to render them useless. The common fault is that the tips are made much too blunt, which, as has already been said, renders it impossible to introduce the higher numbers into the lacrymal sac.

FIG. 10.



Theobald's lacrymal probe.

Although my measurements had convinced me that a probe having a diameter of four millimetres was not out of proportion to the actual size of the lacrymal duct, I had some misgivings as to the practicability of using so large a probe in the treatment of lacrymal strictures; for I feared it might not be possible to introduce it into the sac through the divided canaliculus. I soon discovered, however, that with the tips properly shaped the introduction of a probe of this size was attended by no especial difficulty, and, the better results obtained seeming to warrant it, I found myself, before long, using the No. 16 probe in the majority of cases of lacrymal stricture which came into my hands for treatment. A recent review of my cases, including those met with in children as well as in adults, showed that No. 16 had been used in about sixty-six per cent. of the whole number.

My experience in the use of large lacrymal probes now extends over a period of eighteen years, and with each succeeding year my belief in the value and efficacy of this method of dealing with lacrymal obstructions has become more strongly established. Various theoretical objections to the use of such large probes have been advanced, but they are entirely groundless. It was at first maintained that it is anatomically impossible to pass through the duct a probe having a diameter as great as four millimetres; but this objection was more than met by the data which I had published showing the large normal calibre of the duct. More recently it has been contended that the passage of such large probes must necessarily destroy the physiological action of the drainage apparatus, and that after their use it is impossible that the tears can be carried properly from the

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Fig. 11.

Position of lacrymal probes introduced through lower canaliculus.

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conjunctival sac to the nose.[1] In answer to this I can only say, as I did when the criticism was made, that my experience proves conclusively that the physiological function of the drainage apparatus is not in the least impaired by even the prolonged use of the largest of my probes, and that the tears find their way into the nose, after the strictures have been overcome by this plan of treatment, as perfectly as they do in the normal eye. The introduction of large probes is, of course, attended by somewhat more pain than the passage of small ones, but this is the case only when we are increasing the size at successive probings. As soon as it is no longer necessary to do this, the probe becomes less tight and its introduction causes but little pain,—much less than is caused by the passage of the small probes in the early stages of the treatment. Not infrequently, when a case has been cured by the large probes, the lacrymal canal is left more open than it is in the normal state, the valve-like folds of its walls probably being obliterated by the pressure of the probes, so that when the nose is blown air is apt to find its way through the duct into the corner of the eye; but this causes little or no inconvenience, and is not complained of. One also observes occasionally, and only, it seems, when there is caries of the upper extremity of the duct, a gradual shortening of the divided canaliculus until, in some instances, it entirely disappears, and the probe on being introduced passes directly into the sac, instead of first having to be carried along the canaliculus. This change, however, does not interfere with the passage of the tears into the sac or cause other inconvenience.

My invariable practice, in dealing with obstructions of the lacrymal duct, is to introduce the probes through the *lower* canaliculus (Fig. 11), which I divide well up to its juncture with the sac with Weber's knife, using by preference the pattern in which the beak does not form an angle with the blade. Before introducing the knife, the smallest-sized probes —No. 1, followed by No. 2—are passed through the canaliculus, so as to dilate it somewhat and overcome any stricture which may be present, as this lessens the likelihood of the probed tip of the knife being arrested before it has fairly entered the sac. For overcoming strictures in the lacrymal duct, I prefer not to use a probe smaller than No. 5 or No. 6, and the treatment should be begun with one of these, provided it can be made to enter fairly into the sac. This point assured, any reasonable amount of force that may be necessary to pass it through the duct to the floor of the nose is permissible. Not infrequently, however, a constriction exists at the juncture of the canaliculus and the lacrymal sac which makes it impossible, at the outset, to pass a probe of this size into the sac. Under such circumstances we must be content to begin with one of smaller size,—No. 4 or No. 3. If neither of these can be introduced directly after slitting the canaliculus, it is well to desist from further efforts and allow an interval of two days to elapse, when very often the difficulty previously experienced in finding

[1] Trans. Eighth International Ophthal. Congress, p. 215.

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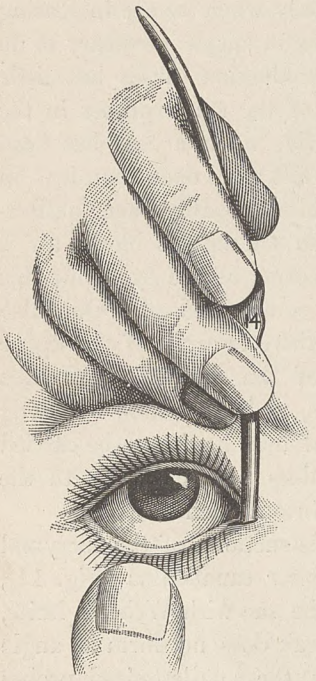
an entrance into the sac will have disappeared. In passing the probe I prefer to stand behind the patient, using the right hand for the right eye and the left hand for the left eye, and keeping the lower lid upon the stretch with the thumb of the other hand. (Fig. 12.)

The existence of a close stricture at the juncture of the canaliculus with the sac constitutes the most troublesome complication that one can meet with in the treatment of stenosis of the lacrymal duct; for it is sometimes impossible to overcome this with the probe, except by turning the probe into a vertical position and forcing its point downward into the duct, a manœuvre which is objectionable for the reason that, though the lid may be kept well upon the stretch to prevent such a misadventure, the probe is liable to make a false passage by passing directly from the canaliculus into the duct without having first traversed the lacrymal sac. The straight, rather sharp-pointed probe shown in Fig. 3 will sometimes be found useful in overcoming this form of stricture. It should be held in a horizontal position and given a drill-like motion—the lid meantime being kept upon the stretch—which causes it to penetrate the stricture. In some cases, however, an entrance into the sac can be obtained only by using a sharp-pointed knife, which should be passed along the slit canaliculus and through the stricture into the sac. The cataract-knife of Sichel I have found very convenient for this purpose.

Fig. 12.



Method of introducing lacrymal probe.

There is a very general belief among ophthalmic surgeons that the use of any force in the passage of a probe through the lacrymal duct is a dangerous and reprehensible procedure, likely to give rise to an impermeable stricture by lacerating the lining membrane of the duct.[1] Abundant experience has convinced me that this belief is entirely groundless. In the vast majority of cases of obstruction of the lacrymal duct the strictures, by the time the cases come to operation, are, in part at least, of bony character, and they necessarily offer considerable resistance to the passage even of a small probe, and still more to that of a large one. In exceptional instances the bony occlusion exists in the form of a thin septum which yields easily to the pressure of the probe, but more often it is thick enough to require decided force to overcome it. Occasionally I have used the strength of both hands to accomplish this (only, of course, in passing

[1] Diseases of the Eye, de Schweinitz, p. 554.

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the larger-sized probes), and I have yet to see any serious consequences result from this procedure. Even should the thin plates of bone which in part form the lower portion of the duct be fractured by the pressure of the probe, it is not a matter of moment, and at most is followed by slight inflammatory reaction and transient ecchymosis. Indeed, I am not only satisfied that the employment of considerable force in the passage of the larger probes is permissible, but I am sure that, instead of doing harm, it has a distinctly curative effect upon the carious walls of the duct, the result being not unlike that produced by the curetting of diseased bone in other parts of the body. Repeatedly, as the result of the use of large probes in this manner, I have observed the rough and carious walls of the duct gradually become smooth and assume a healthy condition, until, finally, the probe on being passed gave the impression of being in contact only with a normal mucous membrane.

The pain attending the introduction of the probe is appreciably lessened, though by no means entirely annulled, by the instillation beforehand of a four per cent. solution of cocaine. It is also well to anoint the probe with an ointment of cocaine and vaseline (ten per cent.). This does not, of course, diminish the pain caused by the passage of the probe, but it does diminish that which is caused by its presence in the duct and by its withdrawal. The introduction of the probe, during the early stages of the treatment, should, as a rule, be repeated every other day,—never oftener, for fear of causing inflammatory reaction, unless want of time compels to such a course. Usually a probe one size larger may be used each time. Occasionally, when the probe is found to be quite loose in the duct, a number may be skipped, and in other cases, when it is exceptionally tight, the same sized probe may have to be passed several times before an attempt is made to introduce a larger one. The probe should be allowed to remain in the duct about fifteen or twenty minutes.

The size of the largest probe which it is advisable to use will, of necessity, vary in different cases. My experience is that it is not only practicable, but that it is expedient, to use No. 16 in about two-thirds of all the cases (including those occurring in children as well as in adults) which one encounters, and I am sure that the cases in which a probe at least as large as No. 13 may not be used with advantage are extremely rare. It may be laid down as a safe rule that in every case as large a probe should be used as can be passed without undue force. Our aim should be, not simply to make a small opening through the constricted portion of the duct (which in a short time will almost certainly become re-occluded), as happens when only a No. 6 or a No. 8 probe is used, but to obliterate entirely the constriction, bring about the complete absorption of the tissue which causes the occlusion, and fully restore the normal calibre of the duct. To accomplish this a No. 16 probe will be required, as has been said, in about two-thirds of the cases, while in the remaining third probes varying in size from No. 13 to No. 15 will be needed. By following this rule we

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shall not only greatly lessen the frequency of relapses, but shall at the same time shorten the period of treatment. My experience with this thorough dilatation plan of treatment now extends, as has been said, over a period of eighteen years, during which time I have employed it in a large number of cases, and have had the opportunity of seeing many of them, from time to time, for years after the discontinuance of the probing, and my observation is that, with a few comparatively rare exceptions, the cases in which the treatment is systematically carried out are completely and permanently cured. So satisfactory have my results been that there is no class of cases which I now undertake to treat with more confidence than I do strictures of the lacrymal canal, and I feel warranted in assuring patients that a complete and permanent cure is in store for them if they will submit to the course of treatment which I consider necessary.

Although in the early stages of the treatment, as has been said, the probes should be introduced usually every other day, as soon as we have reached as large a probe as may be deemed necessary, the length of time between the probings should be gradually increased. At first an interval of three or four days should be allowed to elapse, then a week, then a fortnight, and finally a month, or even two months; and when several of these longer intervals have passed without a tendency to recontraction having manifested itself, the case may with confidence be regarded as cured. Generally it is not difficult, after these long periods of rest, to introduce a probe of as large size as has been previously used, but in exceptional instances a contraction occurs at the juncture of the canaliculus and the sac which makes it difficult or impossible to do this. Including these long intervals, the treatment frequently extends over a period of eight or ten months, but the active treatment, involving the frequent probing, is comprised within as many weeks.

To promote the more rapid absorption of lacrymal strictures the employment of electrolysis has been suggested. This method of treatment, however, has never been generally adopted; perhaps has not received the consideration which it deserves. My own experience with it has not been satisfactory, but is too limited to warrant the formation of a definite opinion as to its value. The negative electrode of a galvanic battery is brought in contact with a probe which has been passed into the duct, while a moist sponge connected with the positive pole is pressed upon the patient's cheek. The chloride of silver ("dry cell") battery is convenient for this purpose, eight to fifteen cells being used. The pain produced is slight, and is experienced chiefly when the current is closed and when it is broken. Cocaine instilled before the introduction of the probe materially lessens the pain.

I have not found it necessary in the treatment of strictures of the lacrymal duct to employ any form of syringe; but, in lieu of this, I prescribe a collyrium, which the patient is instructed to drop into the inner corner of the eye three times a day, after having first emptied the lacrymal sac by gentle pressure with the tip of the finger. The collyria which have

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proved most useful are a solution of bichloride of mercury, varying in strength, in accordance with the sensitiveness of the eye, from 1 to 16,000 to 1 to 12,000, and a solution of alum and boracic acid containing two per cent. of boracic acid and one-half of one per cent. of alum. Weak solutions of sulphate of zinc in combination with boracic acid, and of nitrate of silver, will also at times be found of value. Should undue inflammatory reaction follow the passage of the probe at any time, decided relief will result from the application to the eye, on a pad of gauze or linen, of a lotion of opium and acetate of lead (ext. opii, gr. x–xv; plumb. acetat., gr. xv; aq. dest., oz. iv).

The presence of a lacrymal fistula, even when accompanied by caries of the underlying bone, does not, in my experience, call for especial treatment. The fistula heals promptly, and the carious bone becomes re-covered with periosteum, as soon as the stenosis of the duct has been overcome by the passage of the large probes. Exuberant granulations, if present, should be snipped off or cauterized with nitrate of silver.

The length of time during which the passage of the probe must be kept up varies considerably in different cases, for the strictures yield readily and the inflammation of the sac and of the walls of the duct disappears quickly in some cases, while in others the improvement is comparatively slow. It is never safe to discontinue the probing altogether as long as the epiphora persists or there are any traces of inflammation of the sac. In obstinate cases, however, it is well to increase the interval between the probings, for it sometimes happens that the inflammation is kept up by the too frequent use of the probe. The cases which are apt to prove most stubborn, and in which the prognosis as to complete and permanent cure is least favorable, are those in which the lacrymal affection is but part of a general and severe nasal catarrh. In such cases, and, indeed, in all cases in which the nasal mucous membrane is the seat of catarrhal inflammation, treatment should be directed to the nose as well as to the lacrymal apparatus. The remedy which I have found oftenest of value is bichloride of mercury, the nose being sprayed with a weak solution (1 to 4000 to 1 to 8000), by means of a hand-atomizer, two or three times a day. A small quantity of glycerin and chloride of sodium may be added to the solution with advantage.

Strictures are met with in all parts of the lacrymal duct, the upper extremity of the duct being, perhaps, their most common situation. Multiple stricture is the rule, at least in cases of long standing. The strictures may be circumscribed and annular in form, or ill defined and of wide extent, involving a considerable part of the length of the canal. When a firm stricture happens to be situated at the lower extremity of the duct, the inexperienced operator, and, indeed, in some instances even the operator of considerable experience, may fall into the error of supposing that the point of the probe has reached the floor of the nose, when, in fact, it has been arrested by this deep-seated stricture. Such a mistake as this—a mistake which my experience convinces me happens oftener than it should—neces-

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sarily renders the treatment of little or no avail. If there be any doubt in the mind of the operator as to whether the probe has passed entirely through the duct and has reached the floor of the nose, he should endeavor to dispel the doubt by trying to see the tip of the probe in the nose by means of a speculum and reflected light, or, failing in this, by feeling for it with a bent probe. Nosebleed, which usually occurs when the probe is passed for the first time, is an indication that the floor of the nose has been reached. As the length of the lacrymal duct varies greatly in different individuals, the depth to which the probe penetrates is not always a trustworthy guide as to whether or not it has passed entirely through the duct.

Blennorrhœa of the lacrymal sac, with epiphora, is occasionally met with in infants. The occlusion of the duct in these cases is usually of transient character, being dependent upon a catarrhal inflammation of its walls with consequent engorgement. Operative measures, therefore, should not be resorted to until less radical means have been tried and have proved unavailing. Bichloride of mercury (1 to 12,000), or alum and boracic acid, or a one-quarter-grain solution of nitrate of silver, should first be prescribed, being dropped into the eye, near the inner canthus, after the contents of the sac have been pressed out with the finger-tip. The internal administration of muriate of ammonium, in doses of two to four grains three times a day, according to the age of the child, may also be combined with the local treatment. Should these measures, after a fair trial, fail to effect a cure, the canaliculus should be slit and the duct probed. The strictures usually yield readily, and, as they show less disposition to re-form than they do in adults, it is generally safe, after introducing the probe five or six times at intervals of two or three days, to discontinue the treatment. The administration of a few whiffs of chloroform is necessary when the canaliculus is slit and at each subsequent probing. A case of this character in a child fifteen months old, in which the blennorrhœa and epiphora had appeared within three weeks after birth and had failed to yield to the remedies just mentioned, was recently under my care. Under chloroform the canaliculus was divided and a No. 5 probe was passed without difficulty, two points of constriction being encountered; and subsequently, at intervals usually of three days, Nos. 6, 7, 9, 11, and 12 were successively introduced. A larger probe than No. 12 not seeming to be called for, this size was introduced upon four subsequent occasions, the intervals between the probings being increased to five, seven, ten, and finally, between the last two, to twenty-one days, when the use of the probe was discontinued, the case appearing to be, and, as the subsequent history showed, being, entirely cured.

It occasionally happens that patients from a distance cannot remain under the care of the surgeon for a sufficient length of time to permit the probing treatment to be kept up as it should be to insure the best results. Under such circumstances, after the duct has been thoroughly dilated by the passage of the large probes, it is practicable, as I have found in a number of cases, to teach patients to pass the probes through their own ducts, cocaine

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being first instilled by them to minimize the pain. In this way relapses, which otherwise might occur from the too early discontinuance of the probing, may be avoided. Not every patient, of course, has the self-confidence to do this, but it is not as difficult a feat as might be supposed, and the large size of the probes used under such circumstances practically does away with all risk of a false passage being made.

Although in my early experience in the treatment of lacrymal strictures I used somewhat extensively styles of modern form, sometimes employing those made of silver and at other times fashioning them myself of lead, as recommended by Dr. John Green, the results which I obtained from them were so much less satisfactory than those afforded by the employment of the large probes—the relief being usually only temporary, and relapses after the styles were laid aside being the rule rather than the exception—that I have discontinued their use altogether for a number of years. If a patient suffering with obstruction of the lacrymal duct cannot possibly remain more than a few days under the care of the surgeon, the best thing, in all probability, that can be done for him is to pass as large a probe as practicable, and then to insert a style nearly or quite as large as the probe. For this purpose I have had styles made of aluminium with flattened, curved necks. Their ends are shaped like those of my probes, and they are made to correspond in size with the probes. It is of even more importance to diminish the weight of styles than it is that of probes, for it often happens when a solid silver or lead style is worn for some time that the weight of the style causes the neck to cut into the margin of the divided canaliculus. It will be readily seen, therefore, that there is a distinct advantage in having the styles made of aluminium.

Mr. Zachariah Laurence[1] and others, in intractable cases of stenosis of the lacrymal canal, have recommended removal of the lacrymal gland, asserting that the epiphora and blennorrhœa of the sac are relieved by this procedure. I have had no experience in this plan of treatment; but I am convinced that occasion for resorting to it will rarely present itself, if the strictures of the duct are dealt with after the radical method set forth in the preceding pages.

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[1] Ophthalmic Review, No. 12, p. 361.

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DISEASES OF THE CONJUNCTIVA AND SCLERA.

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DISEASES OF THE CONJUNCTIVA.

DISEASES of the conjunctiva form, on an average, thirty per cent. of all the affections of the eye falling under the care of the surgeon. Their relative frequency, however, varies enormously according to latitude, climate, race, general environment, and condition, being sometimes as low as ten per cent. and often as high as ninety per cent.

The conjunctiva, being a mucous membrane, is subject to the pathological changes to which mucous tissues in general are liable, as well as to some which seem to be peculiar to itself, and, being continuous, through the nasal duct, with the mucous membrane of the upper air-passages, is apt to participate, by continuity, in the inflammatory processes to which the nasal and post-nasal tissues are subject. It is also, on account of the connections of vascular supply, almost without exception implicated to a greater or less degree in the inflammations of the anterior part of the eyeball,—keratitis, iritis, and cyclitis. It seldom takes part in inflammatory affections of the chorioid or the retina.

GENERAL PATHOLOGY.

The conjunctiva being open to inspection, its diseases are more easily studied, clinically and pathologically, than those of mucous membranes hidden from view, and its inflammations have come to serve in a degree as types of mucous inflammations in general.

The most noticeable feature in a conjunctiva in a state of inflammation is the change in its vascularization. In a condition of health its transparency is but little interfered with by the number or size of its vessels, and the white sclera is clearly seen through it. In hyperæmia or congestion this is greatly altered. The vessels are greatly multiplied in number, the veins are more tortuous and increased in diameter, and if there be any considerable amount of exudation the sclera is obscured or entirely shut out from view, the white of the eye assuming a "bloodshot" appearance.

A characteristic feature of this conjunctival congestion is movableness

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of the vessels over the globe: it is in this way, among others, to be distinguished from that deeper ciliary injection, the so-called "circumcorneal injection," confined mostly to the base of the cornea, which accompanies inflammations of the interior of the eye, particularly iritis and cyclitis, and it is often a valuable point in differential diagnosis.

The second stage of the inflammatory process is marked by an abnormal amount of secretion. In a state of health the conjunctival glands secrete only enough mucus for the lubrication of the parts and the maintenance of the proper softness and pliability of the tissues. In the state of hyperæmia there is often a deficiency of secretion, giving rise, in the chronic form especially, to a feeling of dryness and stiffness of the lids. In the acute form of hyperæmia the discharge is almost wholly watery, due to increased secretion of tears. When the second stage of inflammation sets in, there is a hyperactivity of the glands, and the quantity of mucus secreted is increased beyond the normal. When there is no condition leading to pus-formation, this secretion is pure mucus, or catarrhal. When, however, from infectious or other causes, there is formation of pus, pus-cells are found mingled with the mucus, and the discharge is muco-purulent and characterized by a more yellowish tinge of color. In the severer forms pus predominates or constitutes the whole of the secretion. In a pure, uncomplicated conjunctival inflammation there is but little actual pain, because, the tissues being lax, pressure of the inflammatory exudation on the nerve-filaments is slight. What pain there is is never of a pure neuralgic character; the feeling is more one of discomfort and annoyance, heaviness, and heat. The reflex phenomena as manifested by an increased flow of tears (lacrymation) and dread of light (photophobia) are not so marked nor so constant attendants upon conjunctivitis as upon keratitis, iritis, and cyclitis. They may be present during the stage of hyperæmia, but where they are at all prominent or persistent there is cause for suspicion of an implication either of the cornea or of the anterior uveal tract.

The *conjunctivitides* are for general clinical purposes divided into three forms: hyperæmic (and congestive), catarrhal, and purulent. There are many subdivisions, such as croupous, diphtheritic, traumatic, trachomatous, scrofulous, etc., but in these the conjunctivitis is only a symptom or manifestation of a specially operating cause, and each will be treated of under a separate heading.

ACUTE HYPERÆMIA OF THE CONJUNCTIVA.

This may be the initial stage of a catarrhal or a purulent conjunctivitis, or it may exist as an idiopathic condition. It finds its typical representation in an eye into which a foreign body, a cinder for example, has entered. In it we find the vascularity of the conjunctiva much increased, but by an excessive determination of the blood to the part, rather than by its obstructed return. That the arterial circulation is augmented is shown by the larger number of small, straight vessels running towards the cornea.

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The eye has a suffused look, due to more or less increased secretion of the tears. There is usually no discharge of mucus or pus at this stage. The feeling of discomfort may amount in some instances to a distinct pain referable to the eye itself, and if the epithelium of the cornea is involved there may be neuralgic pain, with photophobia.

In this stage it is often a question whether it may not be the beginning of an iritis, and the pupillary reaction should always be carefully noted, though even at this stage of iritis the pupil still remains quite active. If there is a history pointing to the probability of a foreign body, both the conjunctiva and the cornea should be examined carefully by oblique illumination and a magnifier to determine whether one is present. A very minute foreign body embedded in the cornea or conjunctiva, and scarcely visible to the naked eye, will sometimes keep up a hyperæmic condition of the conjunctiva for days or even weeks. The use of the eyes even for a short while is uncomfortable, particularly by artificial light, and the glare of a strong light is annoying.

The suitable *treatment* for acute hyperæmia is, first, the removal of its exciting cause when discernible; and, secondly, assistance in the resolution of the abnormal vascularity. This latter consists in rest and protection of the eye, by a shade or colored glasses, avoidance of smoke, dust, or bright artificial light, frequent bathing in very hot or cold water, according to the feelings of the patient, and instillation of a weak solution (one per cent.) of hydrochlorate of cocaine every three or four hours, according to the severity of the symptoms. In the milder forms, and when the condition is subacute, the cocaine can be combined with a solution of sodium biborate (two per cent.) or other of the milder antiseptics. In cases where there is a suspicion, on account of the severity of the symptoms, of beginning iritis, atropine should be used (gr. ii vel iv ad aquæ, ℥i). In addition to its value in diagnosis the atropine assists in a resolution of the hyperæmic condition.

CHRONIC HYPERÆMIA (PASSIVE CONGESTION) OF THE CONJUNCTIVA.

There are differences other than mere chronicity between this and the affection just described. In fact, we seldom meet with a pure hyperæmia in a chronic state, and what is usually described as such is in reality passive congestion. There is in the latter affection not so much an increased activity of the arterial circulation as a retarded and sluggish venous return. The veins are increased in size, are more tortuous in their course, and often stand out quite prominently on the conjunctival surface. This is much more evident, as a rule, on the palpebral than on the bulbar portion of the membrane. In fact, the bulbar conjunctiva in a large number of cases is not at all affected, though it is always liable to flush up or get "bloodshot" from trivial causes, such as exposure to cold, smoke, loss of sleep, etc.

It is one of the most common of conjunctival affections, and, though not dangerous, is very annoying and uncomfortable, and sometimes renders

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any regular use of the eye impossible. Its frequency arises from the fact that it is symptomatic of or concomitant with many other affections of the eye, the lacrymal apparatus and the upper air-passages, and is rarely absent in the eye-strain of ametropia.

The symptoms on the part of the patient are usually those of discomfort or annoyance rather than of positive pain. There is a sensation of heat, a burning and itching in the eyes, and a heaviness of the lids, with a tendency to keep them closed, particularly in artificial light. There is very commonly a feeling of dryness and stiffness of the lids, as if the eyes were in need of moistening, experienced especially on awaking at night. This is due to a diminution or stoppage of the normal secretion of the conjunctiva, whose office it is to keep the parts pliable and well lubricated. It is for this reason that the condition is sometimes called *dry catarrh*. Very frequently, too, there is a feeling as of grit or sand in the eye, caused by the protrusion of the swollen veins above the level of the conjunctival surface, acting as veritable foreign bodies. These symptoms are always aggravated by causes which bring about an undue determination of blood to the head, such as an attack of coryza, exposure to strong wind, dust, or smoke, late hours, excessive indulgence in alcoholic drinks, etc.

In very few instances does this condition of passive congestion continue for any considerable time without leading to more or less pronounced alteration in the nutrition of the parts, which shows itself by a thickening of the tissue and an enlargement of the papillary structures known as *hypertrophied papillæ*. On the palpebral portion of the conjunctiva the normal smooth appearance is lost and the surface is covered with very minute dots, as though it had been dusted over with the finest meal. These are usually much smaller than the granules which appear in follicular conjunctivitis and trachoma.

Causes.—The condition of passive congestion is very frequently found as an accompaniment of a chronic catarrhal condition of the nasal passages, or as a sequela of the exanthemata or the residuum of some form of acute conjunctivitis. Those employed in a dusty or vitiated atmosphere are most often victims of the affection. Aside from these the most common cause is an uncorrected ametropia or disturbed muscular balance, and even when resulting from other causes it is often kept up by the eye-strain consequent upon these conditions. It is by no means uncommon to find it disappear promptly and permanently on the wearing of properly selected glasses. It is sometimes most obstinate, and will occasionally last for years in spite of all treatment.

Treatment.—The first step in the treatment is to remove the causes which keep up or aggravate the condition. Avoidance of a smoky, dusty, or vitiated atmosphere of any kind is to be insisted upon. If smoking is allowed at all, it should be done out of doors or where the smoke will be readily carried away. The impure air and the glare of theatres or public halls at night are also very detrimental and to be shunned as much as pos-

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sible. When the brilliancy of the sunlight is complained of, colored glasses of blue or of London smoke are to be worn out of doors, and the eyes protected by a shade from direct artificial light. When artificial light has to be used, the best is that of the German student lamp or one of those with an Argand burner giving a steady flame, such as the Welsbach burner. The electric light of the incandescent kind, when it can be properly modulated, should be superior to any other, on account of its steadiness and its near approach to daylight, and the almost complete absence of heat, and in the experience of many is preferred. Still there are some who consider it by no means the ideal artificial light in its present form. Gaslight, particularly from the ordinary burner, is perhaps the worst of all. The correction of all errors of refraction even of low degree is of prime importance, as is also the removal of all disturbed muscular balance. The keeping of late hours and the immoderate use of alcoholic drinks should not be indulged in. Special attention to diet further than a good healthy condition of the general system demands need not be insisted on. The connection in a certain number of these cases between passive congestion of the nasal and post-nasal mucous membrane and the same condition of the conjunctiva being very close, all abnormalities of the nasal passages, such as hypertrophies of the turbinated bones, should be corrected and the catarrhal condition of the nose treated.

Direct medication of the conjunctiva consists in application of some of the mild astringents. The choice among them is very great, and it is often necessary to ring the changes from one to another. The therapeutic indication is to tone up the vascular walls, and a very pronounced irritation is seldom necessary, though occasionally it may be demanded in very obstinate cases. Among the best of the local applications is sodium biborate, ten grains to the ounce of water, a drop to be put in the lower cul-de-sac three times a day. A solution of boric acid, ten grains to one ounce of water, is often soothing to the eye, and can be dropped in three or four times a day. A weak solution of chlorate of potassium is sometimes used for the same purpose. When a stronger astringent is needed, sulphate of zinc, two grains to the ounce, is the best, though weak solutions of sulphate of copper and subacetate of lead can be employed. Sometimes one of these will be found to work well when those efficacious in other cases do not. Equal parts of tincture of opium and water form a useful collyrium. Lately I have found formalin 1 to 2000 very acceptable to some patients. To those conditions of dry catarrh where there is no secretion nitrate of silver is not applicable. This remedy finds its field of greatest usefulness where there is a succulent condition of the conjunctiva with a discharge of mucus or pus. In very obstinate cases it is sometimes advisable to make a profound impression on the vascular walls, which is best accomplished by the solid stick of sulphate of copper applied directly to the mucous surface of the everted lid, the effect to be mitigated by the extent and length of application, and by washing off the surface thoroughly afterwards with water. This may be repeated

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every day or two, or weekly, as the case demands. The alum stick can be used in the same way for the same purpose.

But probably that which contributes most to the immediate comfort of these patients is the spraying of the closed lids with simple water or water mixed with alcohol or bay-rum, or the simple douching of the closed eyelids by the hands with water alone. This can be done every two or three hours or whenever the eyes feel hot or in any way uncomfortable. The opening of the eyes under water, allowing it to come in contact with the globe, is not so efficient, and should be refrained from on account of the swelling of the epithelium which it causes.

In those forms of disease in which the products of tissue-change are not eliminated promptly, and of which *gout* is a typical representative, there is sometimes observed *a tedious form of conjunctivitis*, associated nearly always, after a time, with superficial isolated infiltrations of the cornea, and sometimes with involvement of the underlying sclera. There is not usually any considerable mucous secretion. The attacks continue, with recurrences, sometimes for two or three months, and the exacerbations are commonly associated with changes in the meteorological conditions. In one case I observed some small round deposits in the conjunctiva of the ball. Mere local treatment is ineffective if the diet and mode of life are not regulated. A course of mineral waters, as at Carlsbad or Kissingen, is usually beneficial. Boric acid or sodium biborate solution is the best local remedy.

ACUTE CATARRHAL CONJUNCTIVITIS.

This is the stage of conjunctival inflammation following acute hyperæmia, and is frequently the process of resolution of that condition. The increased secretion of the glands relieves the over-full vessels, and an equilibrium in the circulation is thus restored. But it is sometimes a pathological process, *sui generis*, and due to a specific cause unconnected with a catarrhal condition of the upper air-passages. It is a common accompaniment of epidemic influenza, acute coryza, hay fever, and the exanthemata, and may be idiopathic from exposure to atmospheric changes. It seems probable that these conditions of hyperæmia and congestion only furnish a good field for the activity of some infecting germ. The condition known as "pink eye" is the type of this form of conjunctival inflammation, and the fact that it is frequently epidemic and that it is sometimes highly communicable renders the existence of a specific germ certain. Dr. Weeks, of New York (1886), discovered and isolated a germ, which he found to be a bacillus of a somewhat peculiar shape. This bacillus was noted by Koch in Egypt as early as 1883, but he did not pursue the question further. Confirmatory investigations have since been made by Hansell (1886), Kartulis (1887), and Morax (1894). It is shown in Fig. 1. Weeks made pure cultures of this bacillus and inoculated healthy conjunctivæ with them and produced the disease, the secretion from which, in its turn, produced the disease in other eyes. Others (Axenfeld, Morax, Gifford) have found the

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pneumococcus in the secretion of acute conjunctivitis, and regard it as the pathological agent. This germ is often found in the healthy lacrymal sac, and it is argued that some change in the local conditions renders it virulent.

That acute conjunctivitis is caused by various infectious material cannot be doubted. There are cases of the disease in which in the beginning generally but one eye is affected, where the secretion tends to be muco-purulent, and where there is a more than usual infiltration of the parts, which I have been led to refer to an infection from the discharges of the genitalia of the female. It is most frequently observed in girls from two to six years of age. There seems to be something in the vaginal discharges of the leucorrhœal type, not specific (though some hold that the gonococcus is to be found in all), which is especially poisonous to the conjunctiva. The susceptibility to conjunctival infection of all kinds is, I think, much more pronounced in children and infants than in adults.

Fig. 1.

The bacillus of acute conjunctivitis. (Weeks.) From a photograph of a preparation of the pure culture by Dr. Weeks.

Infection of the conjunctiva undoubtedly takes place also from inoculation of *animal matter* of the kind which is found around slaughter-houses (Despagnet). The inflammation is limited to one eye, and consists of a kind of glandular swelling of the conjunctiva with granulations which exude pus on pressure. There is also more or less swelling of the parotid and cervical glands. The cornea is not usually affected. It is most probably microbic in origin, though the microbe has not been identified. There are few, if any, muco-purulent inflammations of the conjunctiva which are not caused by a micro-organism of some kind.

Under this same heading should be placed the conjunctivitis, which, however, is of the chronic form, which we find associated with obstruction of the lacrymal apparatus, even when there is no purulent inflammation of the sac, the so-called *lacrymal conjunctivitis*. The secretions which should find their exit through the nasal duct are thought by some to be decomposed and form a pabulum for the growth of some of the numerous microbes which enter the eye from the air.

Epidemics of acute conjunctivitis in different localities seem to have different micro-organisms as a cause. Gifford did not find the bacillus of Weeks in cases of epidemic acute conjunctivitis in Omaha, but the pneumococcus; while Morax and Axenfeld found the same in Paris France, and in Würzburg. I have found both the diplococcus of pneumonia and the bacillus of Weeks, generally the latter, in the discharges of acute conjunctivitis in Washington. Gasparini was the first to find (1893) that the diplococcus of Fraenkel and the micrococcus Pasteuri of Sternberg would cause a conjunctivitis of a muco-purulent character. This was confirmed

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by the further investigation of Peters, Bach, and others. The condition of the patient and the general environment play a most important *rôle* in these epidemics. They come suddenly and disappear quite as suddenly, and so many individuals are affected at so nearly the same time as to make a direct contagion from one person to another improbable. With this as with every other micro-organism, a suitable ground for its development is an essential to the full exercise of its pathogenetic qualities. Much as we are indebted to bacteriology in the study of these diseases, we have still a great deal to learn as to the condition under which micro-organisms act with the greatest effect.

Morax (1897) has found a *diplo-bacillus* which he considers the pathogenic factor in *subacute* conjunctivitis, which is contagious. He considers this as quite distinct from the bacillus of Weeks. Peters (1895) also noted the existence of this diplo-bacillus.

All of the microbes found in purulent matter, however, are not obnoxious to the conjunctiva, for the pus of the ordinary hordeolum, or even of acute dacryocystitis, does not give rise, as a rule, to a pronounced purulent conjunctivitis, though there is usually associated with them a hyperæmia or passive congestion of the membrane. There is a form of acute conjunctivitis, or rather hyperæmia of the conjunctiva, found after the insufflation of calomel into the eye while some of the iodides are administered internally. This is the so-called *mercuric conjunctivitis*, and is supposed to be due to the formation of an iodo-mercuric compound from the mercury of the calomel and the iodine in the conjunctival secretion, which is highly irritating. All forms of mercuric application to the conjunctiva should be abstained from during the internal administration of the iodides.

Symptoms, Course, etc.—In the stage of hyperæmia we have the same symptoms and appearances that have been given under *hyperæmia of the conjunctiva*. In idiopathic cases this lasts from a few hours to one or maybe two days. At the end of that time the character of the discharge begins to change. From having been watery it comes to be mucous, or possibly muco-purulent, in quality. This manifests itself first by a gummed-together condition of the lids on awaking in the morning, due to a drying on the lashes of the discharge which has oozed from between the closed lids during the night. On pulling down the lower lid a roll or some flakes of mucus are seen lying in the lower fornix conjunctivæ. In the severe cases which take on a more purulent form small hemorrhages in the conjunctiva are not infrequently observed. There are a slight swelling of the lids and a sense of heaviness and discomfort, though not usually any positive pain, and what pain there may be is confined to the eye and does not radiate to the surrounding parts like the neuralgic pain of iritis, for example. There may be more or less photophobia, but it is not so severe as in keratitis. In many instances, however, the epithelium of the cornea participates, and if to any considerable extent photophobia is a symptom. All these symptoms are more or less pronounced in proportion to the intensity of the in-

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flammation. The secretion varies from a small amount of pure mucus to a quantity of muco-pus which borders on a genuine purulent conjunctivitis, and of course varies at the different stages of the disease. Vision is impaired, partly from a maceration of the epithelium of the cornea by the secretion and from the adhesion of some of the discharge itself to the corneal surface through capillary attraction. In the severer cases the corneal epithelium may be affected, and then there is an exaggeration of the acute symptoms, such as photophobia, lacrymation, and pain. In fact, many ulcerative conditions of the cornea begin in this way, though I do not believe, as some seem to do, that the corneal trouble is necessarily in such cases a complication of the conjunctivitis. In every case it is necessary to examine carefully the condition of the cornea.

Treatment.—Most cases of acute conjunctivitis, when the infection is not strong, go on to recovery with no other treatment than care and cleanliness, and these are the means to be employed in all cases at the beginning. No attempt should be made at an abortion of the process, and leeching is seldom called for. It may alleviate and somewhat shorten the process in strong persons and when the hyperæmia is very pronounced. It would be needless to say that all forms of poulticing are to be avoided, were it not that we not seldom find cases that have been so treated. An application of tea-leaves is, in some parts of the country, a favorite lay remedy for this as well as other eye diseases. The continued application of such poultices leads often to a condition which has been called the "tea-leaf eye," which is characterized especially by a soft, thickened, and macerated condition of the integument of the lids and a matting together of the eyelashes. Nothing should be applied to the eyes which will prevent the speedy exit of the discharge.

The eyes should be cleaned frequently, from every three or four hours to three times a day, according to the amount of the discharge, with an aseptic or mild antiseptic solution, among which boric acid, ten grains to one ounce of water, is perhaps the best in acute cases. This is best done by emptying the contents of an ordinary eye-dropper two or three times on the eye held open with the fingers. Cloths saturated with lead-water and laudanum, or simply wrung out of cold water, may be laid on the closed lids for their cooling and soothing properties. The douching of the eyes with cold water or the use of the spray often brings relief to the feeling of heaviness and heat. If the hyperæmia seems to be in excess, bathing the eyes frequently in water as hot as it can be borne will have a soothing effect. To prevent gumming of the lids together during the night the edges should be anointed with vaseline at bedtime, or, better still, with an ointment of the yellow amorphous oxide of mercury, one grain to two drachms of cosmoline.

When the secreting stage sets in, the use of the mild astringents should be begun. The best of these is sodium biborate, ten grains to the ounce of water, a few drops in the eye three or four times a day. Later the more

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purely astringent remedies, such as sulphate of zinc, two grains to the ounce of water, night and morning, are indicated. Since I have become acquainted with formalin, however, I have used this almost exclusively, in solutions varying from 1 to 1000 or 1 to 2000 every four hours, even in the early stages. If the purulent character is at all prominent, a weak solution of nitrate of silver, gr. ii ad oz. i, is to be preferred if the formalin in the stronger solution of 1 to 1000 has not been effective. These drops may be applied by means of a dropper or camel's-hair brush to the lower cul-de-sac three times a day, the interval to be lengthened as the discharge lessens to once a day. In cases where the cornea is implicated atropine should be used in addition to the other means recommended. When there is an ulcer or abrasion of the cornea a lead lotion is contra-indicated, lest a deposit of lead in the cornea take place. As the discharge is more or less infectious, the cloths, towels, and other articles used about the eyes of the patient should not be used by others, and as much isolation as practicable should be insisted on. Protective spectacles should be employed against bright and glaring lights. Smoking should be entirely prohibited or much restricted, and alcoholic drinks used very sparingly, if at all. For the subacute form in which the diplo-bacillus is the exciting cause, Peters and Morax have found the sulphate of zinc solution almost a specific. The End

PURULENT CONJUNCTIVITIS.

The conjunctival inflammations hitherto considered are attended with discomfort or even pain, and entail a restricted use of the eyes temporarily, but they are not liable to lead to a permanent impairment or loss of vision.

So much, however, cannot be said of the purely purulent forms of the disease. A very large part of the blindness in the world is due to the ravages of purulent conjunctivitis, variously estimated from one-third to one-half. (See article on "Blindness," vol. ii. of this System.)

It is essentially an infectious disease, and its most frequently producing cause is a diplococcus discovered by Neisser in 1879 as distinctive of the discharge of gonorrhœa. There can be no doubt, in the light of recent investigations, that other microbes aside from the gonococcus give rise to a purulent inflammation of the conjunctiva. Stephenson (1895), among others, has found the staphylococcus pyogenes aureus and albus and the streptococcus pyogenes, and, as stated under Acute Conjunctivitis, the pneumococcus is found in the muco-purulent discharge in that disease. Axenfeld, Morax, and others have found the pneumococcus in the discharge of ophthalmia neonatorum, and it may not be without significance in this connection to state that it has also been found in ulcer of the cornea with hypopyon. It would seem that the condition of the conjunctiva at the time is largely the controlling factor as to what micro-organism is led into activity. The gonococcus is very tenacious of life, and often when the urethral or vaginal discharge has seemingly lost all purulent characteristics or apparently ceased, there may be some cocci still concealed in the folds of the mucous

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membrane which when transferred to the conjunctiva find there a suitable ground for their more rapid propagation. Infection of the eye may be brought about in a number of ways, but generally it is by the patient's own hands. After manipulation of the genitalia, the hand (the right one in those who are right-handed) is used to rub the eye or the face about it, and the *materies morbi* finds its way into the conjunctiva. It is often possible to tell whether the patient is right- or left-handed by the eye which is first affected. The infecting material may be also carried by the towels, handkerchiefs, and dressings used by a person affected with gonorrhœa or with purulent conjunctivitis. I once saw a chicken with its conjunctiva infected from some dressings from the eyes of a child suffering from ophthalmia neonatorum which a careless nurse had thrown into the yard instead of into the fire as she had been directed. For this reason physicians, nurses, and attendants should always exercise the utmost care in treating both gonorrhœa and purulent conjunctivitis, and all patients suffering from this form of conjunctival disease should be isolated from other patients when possible. As the morbid discharges from the vagina are also very dangerous to the conjunctiva, the same scrupulous care should be extended to the treatment of diseases of the female genitalia. It would seem from some reports that it is possible for the infecting material to be carried by the air. The probability of this, however, is very scant. There have been cases reported where the infection was carried by the urine of a person affected with gonorrhœa, who had used it to wash the eyes affected with a simple conjunctivitis, a treatment not uncommon among some of the lower orders.

There may be quite a quantity of pus discharged from an eye affected with an inflammation beginning as a pure catarrh of the conjunctiva. This may not be infective to the same degree nor so dangerous to the integrity of the eye as the specific form. In the beginning, however, only a microscopical examination of the discharge can establish the differential diagnosis. There seems to be good reason, too, for believing that there may be a conjunctivitis, not necessarily suppurative, caused by the toxines of the gonococcus circulating in the blood, and not due to external infection, analogous to the gonorrhœal iritis which is sometimes met with.

Symptoms.—The first manifestations are those of acute hyperæmia or catarrh, and they may commence in from ten hours to three days after infection. The period of incubation as well as the virulence of the disease varies, in a measure at least, with the intensity of the poison. In the severer forms the violent symptoms develop very early and rapidly. The lids become red and swollen and the integument smooth and somewhat hard and glistening. There is a rather abundant discharge of thin fluid, which is apt to contain some flocculent material. It is sometimes reddish from the presence of blood-corpuscles. The eye feels hot, and there is usually some rise in the general temperature of the body. If the lids can be separated so as to allow of an inspection of the eyeball, the conjunctiva will be seen to be much infiltrated with serum, its vascularity is increased, and

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there may be some hemorrhage in its substance. But soon this gives place to the second stage, in which a copious purulent discharge is the chief feature. The pus is thick and creamy, is formed in large quantity, and is constantly oozing from the palpebral fissure. There is usually at this stage some subsidence of the hardness of the lids, but the swelling of the conjunctiva is not diminished. It mounts up around the base of the cornea (chemosis) and overlaps its edge, causing it to appear sunken deep below the surface of the swollen conjunctiva. The pain and heat usually abate somewhat with the establishment of a free discharge. The lid swelling in some cases is so great that the upper lid hangs like a bag down over the lower one and it is not possible by manipulation to expose the cornea to any extent. Every effort should be made, however, to inspect it, for the great danger of the disease lies in its involvement. This corneal complication may come from two sources. Its epithelium and anterior layers become macerated and destroyed from constant contact with the purulent matter, allowing the microbes to penetrate the corneal tissue and multiply with destructive rapidity. For this reason it is necessary to avoid making any abrasion of the cornea with instruments used in cleansing or examining it. The corneal implication is most apt to occur first at the edge under the overlapping chemosis. It manifests itself as an ulceration, sometimes with clear outlines, but usually with yellow edges, and is apt to extend with rapidity, involving in time the whole tissue. In other instances the tense swelling around the base of the cornea causes such pressure on the channels of nutrition as to bring about its sphacelation as a whole. The inflammation of the capsule of Tenon, which undoubtedly exists in some cases, also assists in this process. The epithelium becomes steamy, and the tissues seem to melt down like snow under the sun. Here the entire anterior layers of the cornea are destroyed, and only some portions of the posterior layers, including the membrane of Descemet, remain, through the defects in which at various points the iris protrudes as little knots, the so-called *staphyloma racemosum*. Sometimes the destruction is so extensive as to allow the lens to be expelled, and occasionally there is a panophthalmitis, probably due to an entrance of pyogenic germs into the interior of the eye. This panophthalmitis may occur after the purulent discharge from the conjunctiva has ceased, the microbes which entered through the perforation in the cornea remaining dormant for a time. I saw one case in which the panophthalmitis began three weeks after the healing of the corneal ulcer. The substance of the conjunctiva itself is not often destroyed or ulcerated to any considerable extent, though it rarely fails of having some scar left as a reminder of the terrible battle it has passed through. If left to itself, the disease runs its course in from three to six weeks, generally ending in a chronic inflammation attended with a thickening of the membrane and a slight muco-purulent discharge. The conjunctiva is left in a condition which renders it very susceptible to the development of other diseases, such as trachoma and the diphtheritic and croupous forms of conjunctivitis.

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Treatment.—The most satisfactory treatment of the disease is prophylaxis, and whenever possible such patients should be isolated. When one eye only is affected, the other should be hermetically sealed by a bandage, the Buller shield, or other device of a similar kind. No risk should be run of a possible infection of other eyes. When there is a suspicion that some infecting matter has gotten into a healthy eye, as from syringing or from the use of infected handkerchiefs or towels, etc., a drop of a one or two per cent. nitrate of silver solution should be applied to the everted conjunctiva at once. Nitrate of silver has the power more than any other drug we possess of neutralizing this poison. Formalin, however, on account of its penetrating property, promises to be very useful in this field. Or, if this seems too harsh a treatment for a mere suspicion, the eye should be at least washed out thoroughly with an antiseptic solution of mercuric bichloride or a saturated solution of boric acid or formalin 1 to 1000, and watched carefully for two or three days.

Any attempt at abortion after the stage of hyperæmia has once set in and before that of secretion begins is to be deprecated. In fact, nitrate of silver or other caustic at this stage of the affection seems to aggravate it, though it is a method of treatment which has not been without strong advocates. During this period, which is, however, usually short, and during which the patient is seldom seen by the surgeon, the treatment for the hyperæmia laid down elsewhere is applicable,—namely, mild antiseptics, cocaine, and bathing in hot water. If the patient is not anæmic, three or four leeches may be applied to the temples and the bleeding encouraged by warm applications. Scarification of the conjunctiva at this stage is not followed by much benefit.

It is commonly when the stage of secretion has already set in that the patient is first seen by the surgeon, and at this period the sovereign remedy is nitrate of silver. For the discovery of this almost specific in the treatment of purulent conjunctivitis we are indebted to the genius of von Graefe, though its mode of action is doubtless somewhat different from that which he suggested. While its power as an antiphlogistic in the conventional sense is undoubted, its greatest influence is probably exerted as a destroyer of the infecting germs. As a germicide nitrate of silver stands in the first rank, and it has this power in addition, that it destroys the epithelial layer of the mucous membrane and with it the germs which there find an abiding-place.

It is not necessary, however, that it always be used in the strong solutions which have been recommended. When the secretion is not very great in amount and the swelling not excessive, a solution of from two to five grains to the ounce of water may be brushed over the surface of the conjunctiva, after it has been cleansed of the overlying secretions, two or three times a day. But when there is great swelling of the lids and the discharge is correspondingly increased in amount, the strength of the solution may be ten, twenty, or even, in cases of malignant severity, forty grains to the ounce. These strong solutions should not be used oftener than once a day, and then

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should be immediately followed by ice-cold applications to the lids in order to diminish the amount of reaction.

The effect of these strong solutions or the mitigated stick (composed of equal parts of nitrate of silver and nitrate of potassium) should be limited to the parts to which they are applied by immediately washing the surface off with salt and water, thus neutralizing the excess of silver by forming silver chloride. The object of these severe applications, it was formerly supposed, was to form an eschar on the conjunctival surface, which would stop more or less for a time pus-formation. As we regard it now, however, these stronger applications act through their more intense anti-germicidal power. When this white eschar is thrown off the pus-secretion begins again, and it is then time to make another application, whose effect can be regulated by the amount of time it is allowed to remain on before being washed off. The intensity of effect must be regulated in accordance with the amount of the discharge. The time required for the casting off of the eschar varies from six to eight hours in very severe cases to twenty-four or thirty-six hours in the mild ones. Recently formaldehyde or formalin [1] has come into use as an antiseptic, and my own experience with it has been very satisfactory.[2] It may not supplant nitrate of silver in the acute stages, but in the later stages it can be used in the strength of 1 to 1000 or 2000 as a cleanser. In the strength of 1 to 500 its effect is about that of silver of two per cent. Permanganate of potassium, which is being used with such good results in gonorrhœa of the urethra, has been highly recommended by some writers as a local application in purulent conjunctivitis. The strength should be from three to ten grains to one ounce of water, and it should be applied thoroughly to the conjunctival surface twice a day in severe cases, and once a day in the milder attacks.

Only second in importance to the nitrate of silver treatment is the proper cleansing of the eye. In fact, in very mild cases keeping the eye thoroughly clean with an aseptic liquid will sometimes suffice for a cure; while without the strictest attention to cleansing all other treatment is liable to be ineffectual. This cleansing is done with a mild sublimate solution (1 to 5000 or 10,000) or a saturated solution of boric acid or formalin 1 to 2000. It is not always easy of accomplishment, especially when the lids are greatly swollen. The lids should be opened with great gentleness as widely as possible by the fingers and thumb of one hand, and the liquid, previously warmed, squeezed from absorbent cotton and allowed to run over the exposed secreting surface. An endeavor should always be made to reveal as much of the conjunctiva as possible and to remove all the secretion from among the folds around the cornea. This cleansing should be made with a frequency corresponding to the amount of the discharge, varying from every ten or twenty minutes to three or four times a day. In severe cases two

[1] Formalin is a forty-five per cent. solution of formaldehyde. Formaldehyde itself is a gas and a strong escharotic.

[2] The Use of Formalin in Ophthalmic Practice. Ophthalmic Record, March, 1896.

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attendants are required, one for the day and one for the night, for the attention must be unremitting.

It is not wise to use a syringe for the removal of the secretion, for, in addition to the possible danger of injury to the cornea by the awkward manipulation of the instrument, there is the greater one of throwing the matter from the eye of the patient into that of the attendant. An apparatus for irrigating the conjunctival sac has been devised by Dr. Andrews of New York. It consists of a hollow lid-holder with perforation at its free edge. This is introduced under the lids and an antiseptic fluid sent through it by means of a siphon-tube.

For the purpose of keeping the inflammation in check, ice-cold applications to the lids are undoubtedly of great utility. The application of cold to the eye is best accomplished by means of cloths which have been lying on a block of ice. There should be a number of them, in order that a change can be made as soon as the one in use becomes warm, which is generally in a minute or two. This must be kept up day and night if we wish to retain the beneficial effect of the cold. For this reason it is necessary to have a day nurse and a night nurse. The cloths should not be larger than will well cover the eye, and should be of sufficient thickness to retain the cold without being heavy on the sensitive eye. Small bags filled with pounded ice are also used. These take longer to prepare, but also last longer.

It is contended by some that the cold hinders and retards the development of the infecting germs, but this seems hardly possible with a cold which is not intense enough to affect seriously the vitality of the parts.

Some good authorities rely upon cold applications greatly, but it must be evident, when we regard the pathological conditions, that their indiscriminate use cannot always be beneficial. In the beginning, when hyperæmia is still a prominent feature, the continuous application of cold must, by diminishing the blood-supply, act as a positive antiphlogistic and thus lessen the intensity of the process. But when the stage of secretion has once been established this action is no longer demanded, and is, in fact, on some accounts contra-indicated. The tissues are infiltrated with exudation; there is stasis of the venous system, with atony of the vascular walls. For the relief of these continuous cold cannot act to the best advantage. On the contrary, what is needed is a temporary hyperæmia which shall increase the activity of the circulatory and absorbent systems, and this can be accomplished better by the periodical application of heat. Dr. Leartus Connor, of Detroit, was the first to advocate this method as a substitute for cold in all stages of the disease. He and others have reported very satisfactory results from its employment, and in my own practice I have been pleased with it. It is certainly in keeping with the theory and practice of treating suppurative inflammations elsewhere. Moreover, there is one thing more important than all the rest, which must be continually kept in mind in our management of these cases, and that is the integrity of the cornea. We must never forget, in our therapeutic endeavors, that we are

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fighting not for the conjunctiva, but for the preservation of the cornea, and everything should be avoided which tends to diminish still further the vitality of a tissue whose nutrition is already so much impaired. When the corneal surface begins to have a steamy look, or there are points of commencing ulceration, then certainly is the time to abandon cold and resort to heat. The application of heat is best made by immersing the eye in a cup or tumbler filled to the brim with water as hot as can be borne. It is easily done by holding the cup in the hand and bending the head as first suggested by Dr. Connor so as to bring the eye and the surrounding parts into the water gradually. This is cleanly and convenient of application, and so thoroughly fulfils the requirements that no other method need be mentioned. In the case of children and those not able to assume the erect posture hot borated fomentations can be used.

This bathing may be continued for some minutes, and repeated, according to the intensity of the symptoms, every one, two, three, or four hours.

But, in addition to cleanliness, the use of nitrate of silver, cold, and heat, there are other proceedings which are employed to facilitate the resolution of the process and to lead it to a favorable termination with the least injury to the cornea. Among these is a scarification of the swollen and tense conjunctiva. In the early stages this may be of more or less benefit in unloading the vessels, but when the infiltration has once taken place the relief to the tension is usually insignificant. A diminution of pressure is much better accomplished by a division of the outer canthus (canthotomy). The incision should be made out to the edge of the orbit and include the orbicularis muscle. It is usually best effected by means of a pair of strong scissors. This not only lightens the pressure on the ball, but enables the eyelids to be more easily everted for cleansing and for the application of the nitrate of silver. Recently vaseline has been recommended for use as a cleanser, and it is thought to have likewise some curative properties. A quantity of pure white vaseline is taken on a probe and introduced as thoroughly as possible under the lids from one to four times a day.

When the cornea has become involved it is questionable whether any treatment directed specifically to it is of any avail. So long as it is bathed in pus, local applications can have but a transitory or no effect. Sulphate of atropine may be used for the corneal ulceration, and eserine for the purpose of lowering the tension and contracting the pupil and thus limiting the prolapse of the iris, especially if the ulcer is at the periphery. In such cases I have recently used formalin 1 to 60, with which the ulcer is touched once a day. Formerly I used finely powdered iodoform with good effect. One thing, however, that has been recommended should never be done, and that is puncturing or snipping off of the prolapsed iris. This is almost certain to bring about a state of affairs we wish above all things to avoid. So long as the iris is intact it opposes an effectual barrier to the entrance of germs into the interior of the eye, but when it is cut or perforated they can enter more easily and entail a panophthalmitis.

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After the purulent discharge has measurably or altogether ceased, there remains a swollen or hypertrophied condition of the conjunctiva which sometimes persists for a long while. To this state nitrate of silver is not so applicable. It is here that the more purely astringent remedies are most useful, and at the head of these stands sulphate of copper. A smooth crystal of this should be applied to the conjunctival surface, particularly to the very much enlarged retrotarsal folds, every other day, the excess of the copper being washed off with water. An alum stick may be used for the same purpose, or a solution of tannin in glycerin. A snipping off of the enlarged retrotarsal folds which sometimes protrude from between the eyelids is not to be commended as a routine practice.

OPHTHALMIA NEONATORUM.

There is a form of purulent conjunctivitis which is so distinctive in its origin, so disastrous in its results if left to itself, and so important as to its specific prophylaxis and treatment, that a separate consideration for it is demanded. This is the purulent conjunctivitis of the new-born infant (ophthalmia neonatorum).

At a time varying from a few hours to several days after birth a redness and swelling of the lids of the infant, accompanied with a discharge more or less purulent, mark the beginning of this disease. These symptoms increase usually with great rapidity, and soon instead of eyes there appear in the sockets what seem to be two enormous abscesses, from which there issues an almost continuous stream of pus. We have here essentially the same clinical picture that we have drawn in the preceding section for purulent conjunctivitis in the adult, only, as a rule, it is greatly exaggerated in all its features.

The swelling is more pronounced on account of the greater laxity of the tissues in infancy, and this makes an inspection of the cornea in severe cases impossible. The amount of pus secreted is sometimes enormous. Whenever the lids are pulled apart pus pours out almost by the teaspoonful. All cases, however, are not of this intensity, and the severity ranges from the most virulent down to a conjunctivitis of hardly more than a mild muco-purulent type. It should not be forgotten, however, that all cases begin in essentially the same manner. The longer after birth the disease commences the less severe the attack is likely to be. A mild form of catarrhal conjunctivitis quite frequently appears two or three weeks after birth, but this, though sometimes obstinate, is rarely specific and is not dangerous.

Like other forms of purulent conjunctivitis, it, too, is one of infection, and, except in rare cases, from one definite source, the vagina of the mother. The infection takes place during the passage of the head through that canal in the process of parturition, though some have thought it possible for the infection to have taken place *in utero*. There may be other methods of infection, such as have been indicated in treating of the etiology of purulent

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conjunctivitis of adults, but from the conditions surrounding the infant these must necessarily be very rare. The idea that any severe conjunctivitis can be caused by exposure to bright light, catching cold, washing with strong soap, etc., cannot now be entertained by any one in the light of our recent knowledge regarding the prophylaxis and treatment of the disease.

It is not contended that every case of ophthalmia neonatorum is one of gonorrhœal conjunctivitis. That is a thesis which it would be difficult to maintain, for a careful examination of the discharge in a number of cases has failed to show the presence of the characteristic gonococcus. As has been stated before, all discharges from the vagina of the female which are pathological have the property of setting up a purulent inflammation in the conjunctiva, and the conjunctiva of the infant seems peculiarly liable to be thus affected. For virulency and destructiveness the gonococcus does not seem to be essential, but its presence should always be suspected and looked for.

In quite a number of cases of the less severe form, the pneumococcus has been found in sufficient numbers to warrant the suspicion of cause and effect (Parinaud, Morax, Gasparini).

That the infection comes from the vagina of the mother, almost without exception, is proved by the measures that have been instituted for its prevention and cure, the success of which is one of the greatest triumphs of modern scientific medicine.

This is one of those diseases for which it can be claimed that absolute prevention is possible, and with proper precautions there need never be another case of destructive ophthalmia neonatorum. What that means we can more fully understand when we call to mind that more than one-quarter of all the blindness of the civilized world at the present time is caused by this disease (Magnus, Fuchs, and many others). Not only that, but the blindness, beginning as it does with life itself, so handicaps the person affected that instead of becoming a producer he remains, in all but a small number of cases, during the remainder of his life a consumer only and a charge upon society. The loss to the common wealth of the community from this cause is enormous, as a little calculation will show. There were in the United States, according to the last census, more than fifty thousand blind persons. This we may properly conclude to mean those totally blind, and not those only partially so. Considering, then, the insufficiency of the census reports, which are always under rather than over the actual number, and the average percentage of the blindness caused by ophthalmia neonatorum as more than twenty-five per cent. (it varies from fifteen per cent. to fifty per cent. in various statistics), we are certainly within the mark in assuming that fifteen thousand of these blind persons lost their sight from neglected sore eyes in infancy.

Taking the cost of maintenance of a single person, according to the statements of our best regulated asylums, as, at the least, one hundred and thirty-two dollars a year, and the average net earnings of a single able-

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bodied person as one dollar a day, we find that the total loss to the common wealth of the United States from the ravages of this disease reaches the sum of seven million five hundred thousand dollars annually. Then there are the moral and humanitarian aspects, which are so apparent as to require only a mention to have their importance realized. It is, unfortunately, not asserting more than statistics amply warrant to say that had proper measures been instituted at the right time not one of those fifteen thousand cases of blindness would have occurred.[1]

The disease being one essentially of infection and from a well-recognized source, its *prevention* is of practically absolute certainty. The honor of having demonstrated this belongs to Professor Credé, of Leipsic, who first brought it before the profession in 1882. Previous to the introduction of his methods the percentage of ophthalmia neonatorum in cases of parturition ranged, in various lying-in institutions, from 19 to 4. Now in those institutions where it is promptly and thoroughly carried out it does not exceed 0.2, and even this can usually be attributed to some neglect or carelessness on the part of the attendant. In Widmark's statistics on ophthalmia neonatorum (1895) in Stockholm, where Credé's method was early introduced and its use rapidly diffused, the decrease in the percentage of the disease fell in the general clinic from 1.2 in 1884 to 0.24 in 1890. And whereas the corneal complications in the cases that did occur were thirty per cent. in 1884, they were only six per cent. in 1888. In the last statistics published by Kösling (1896), among 17,767 cases in which no preventive measures were used there was 9.2 per cent. of ophthalmia neonatorum; in 24,724 in which a 2 per cent. nitrate of silver solution was used, 0.65 per cent.; in 1223 cases treated with a 1 per cent. nitrate of silver solution, 2.24 per cent.; in 965 treated with 1 per cent. sublimate solution, 0.6 per cent.; in 1396 treated with other sublimate solutions, 0.4 per cent.; in 701 treated with iodine trichloride, 1.2 per cent.; in 6155 treated with sterilized water, 2.8 per cent.; in 1623 treated with carbolic acid, 7.7 per cent. The disadvantage of the sublimate solutions is the greater irritation they cause. The procedure is very simple. As soon as the head has passed the vulva, the face of the child is wiped clean, the eyelids are opened with the fingers, and a drop of a two per cent. solution of nitrate of silver is let fall from a glass rod on the eyeball. The reaction that follows from this is usually so slight as to require no interference, but occasionally it is severe enough to call for mild antiphlogistic measures, such as cold cloths and cocaine and atropine. At least one case of severe hemorrhage from the conjunctiva has followed the application, but any such accident is very rare. In all only four cases of disastrous results after the use of this method have been reported up to the time of this writing; also one after the use of sterilized water.

[1] This sweeping statement has been criticised in some quarters. It is asserted that ophthalmia neonatorum has appeared after all the precautions we have mentioned have been taken. Such cases are certainly very uncommon, and are only the exceptions that are recognized to exist to all general rules. Theoretically, the above statement is true in full.

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This treatment appears unduly harsh in the opinion of some, and these are not willing to adopt it in all cases, and in private practice various circumstances might make it difficult to carry it out as a routine proceeding.

The least that can be expected, however, of the accoucheur under all circumstances is that he give close attention to the eyes, have them carefully cleansed with an antiseptic solution and closely watched for the appearance of any symptoms of inflammation, and when these show themselves attack the disease very vigorously at once.

It has been thought that simply attention to the vagina previous to and during parturition would suffice. A thorough washing out of the vagina with an antiseptic fluid regularly, some days before expected confinement, has been advised, but it has not been found so efficient as the Credé method. It is next to impossible to render a septic vagina completely aseptic. However carefully the irrigation is done, there are likely to remain some germs hidden away among the folds of the mucous membrane. For absolute certainty the plan of Credé is the best that has yet been discovered, as well as the simplest. Other antiseptics have been recommended, as sulphocarbolate of zinc, mercuric bichloride, the dusting of iodoform in the conjunctival sac (Tarnier), etc.; but none of these are as certain in their effects as nitrate of silver. And it is highly gratifying that since a knowledge of this method has been disseminated among the profession wherever statistics have been taken there has been a noticeable falling off of the disease and its results, such as staphyloma, corneal opacities, etc. It is, therefore, the manifest duty of ophthalmic surgeons and all good citizens to use every means in their power to extend among the laity, and especially among the poor, in whom the disease is most rife, a knowledge of the danger of an affection which the ignorant, both professional and non-professional, have been wont to regard as simply "a cold in the eyes" which a little breast-milk would suffice to cure.

This is a matter which in an important degree concerns the state, and is very properly a subject for legislation. Most of the European governments require by law the reporting of all cases of ophthalmia neonatorum to the constituted authorities, and up to the time these lines go to press the following States in this country have passed laws making the report of cases of ophthalmia neonatorum compulsory,—viz., Maine, New York, Rhode Island, Minnesota, Ohio, Maryland, Connecticut, Missouri, Iowa, New Jersey, Pennsylvania, and South Carolina. This includes a population amounting to about thirty-five million inhabitants. The American Medical Association, on the recommendation of the Ophthalmological Section, has given the sanction of its approval of the Credé method in all public institutions. It is hoped that medical men and others who are interested in the welfare of the community will keep the matter in agitation until each State or the Congress of the United States shall have enacted laws which will bring all cases of ophthalmia neonatorum, as soon as the disease manifests itself, under the care of a person competent to treat it. It

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is to be regretted that the government of Great Britain has persistently refused to move in the matter.

In addition, however, to general legislation, it is necessary that the laity be thoroughly informed of the dangers of a disease which they have been accustomed hitherto to regard as simple and harmless in its nature. All charitable institutions or organizations, whether of church or of state, which have to do with women likely to become mothers, or with those having the care of children, should make it a point to keep them informed of the necessity of a careful watching of the baby's eyes, and for this purpose should have on hand for free distribution cards something like the following:

"When the baby's eyes begin to look red and to run matter, take it at once to a doctor. It is very dangerous, and if not treated properly one or both eyes may be lost."[1]

We should insist, too, upon the teachers of obstetrics in our colleges dwelling more strongly upon the dangers of the disease and pointing out more specifically the means for its prevention and of treating it when established. In only a few colleges in the United States, I believe, is this done, though the obstetrician has within the last few years, thanks to the teachings of Credé, been more alive to his duty in the matter than formerly, and is now beginning, both in his lectures and in his text-books, to realize the importance of the disease, the prophylaxis of which belongs to him rather than to the ophthalmic surgeon.

In the *treatment* of the disease the same principles hold as for purulent conjunctivitis of the adult. The tender age of the patient is no bar to the most energetic treatment.

Constant attention, cleanliness, with aseptic or mild antiseptic solutions, heat or cold, and nitrate of silver are the means to be relied upon here as there, and as the swelling of the lids is usually greater in the infant the division of the outer canthus is more frequently called for. After the subsidence of the acute symptoms there usually remains a chronic inflammation with much hypertrophy of the conjunctiva, which may persist for many weeks. As the discharge begins to lessen in quantity and lose its purulent character, the dosage of the nitrate of silver should be gradually diminished, and one of the pure astringents, such as zinc sulphate gr. ii ad oz. i, alum, or copper crystals, substituted when its purulency has disappeared.

The corneal complications of purulent conjunctivitis are subject to the same rules of treatment as the same corneal affections from other causes, though, from the circumstances of the case, with less hope of benefit. The caustic treatment of serpiginous ulcerations, for instance, can hardly be effective here in the general run of cases, because, the source of infection lying outside of the ulcer, it cannot be removed by any treatment directed solely to the ulcer itself. At this stage of the disease formalin is of especial benefit both to the conjunctival and to the corneal affection, and may be used in

[1] Used in the Sheffield (Eng.) General Infirmary.

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the strength of 1 to 1000 or 1 to 500. It is only when the suppuration has ceased or become much ameliorated that we can hope for a stay of the destructive ulceration by this or other means. But when the ulcer is not large and is only beginning, such treatment is strongly indicated. (For fuller details of pathology and treatment of corneal ulceration in purulent conjunctivitis, see chapter on Diseases of the Cornea.)

MEMBRANOUS CONJUNCTIVITIS (CROUPOUS, DIPHTHERITIC).

In the ordinary forms of muco-purulent or purulent conjunctivitis a thin layer of yellowish-white material is often seen in patches on the surface of the conjunctiva. This is nothing more than degenerated epithelium mingled with pus-cells, and is easily washed or wiped off.

There is a form of conjunctivitis, however, in which the formation of what appears to be a distinct membrane is the important feature so far as it indicates an exudative form of inflammation, and, almost certainly, a specific cause. It has been thought possible to divide this so-called membranous conjunctivitis into two distinct forms,—the croupous and the diphtheritic.

There appear, indeed, to be ample grounds for making two forms of membranous conjunctivitis, so far as regards their clinical aspects; but there seems to be some difficulty in classifying these categorically as croupous and diphtheritic on the basis of either etiology or bacteriology. The same difference of opinion exists here as in regard to the essential nature of croupous and diphtheritic angina, some holding them to be identical in origin and differing only in intensity of manifestation, while others believe them to be distinct affections with independent causes. The so-called croupous form is sometimes called pseudo-diphtheria. It would seem that the presence or absence of the bacillus of Klebs-Loeffler cannot be relied upon certainly in the differential diagnosis of diphtheria, for, as the investigations of Tarze, Uhthoff, Sourdille, Morax, and others have shown, it has been found in cases which clinically fall in the croupous group, and its presence has not been demonstrated in severe cases which have all the characteristics of diphtheria, as has been shown by Weeks,[1] by Standish, and in my own experience. The only microbe found in these cases was a streptococcus. We should, however, still hold our opinions plastic in regard to the question, on which additional light is being constantly thrown by investigation and experiment. It is possible that the absence of the Klebs-Loeffler bacillus in cases of pronounced diphtheria from a clinical point of view may be due to the destruction of the bacillus by its own toxine; and it is very easy to understand the implanting of a streptococcus infection on a diphtheritic soil. In fact, at the time these lines go to press there is a probability of the existence of a membranous conjunctivitis of a purely streptococcus origin.

[1] New York Eye and Ear Infirmary Reports, January, 1895.

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For clinical purposes, therefore, it is still best to retain the distinction and treat of each form separately, though we are not without hope that in the near future we shall be able to classify them definitely according to the bacteriological findings.

Croupous Conjunctivitis.—The onset of this form is very like that of purulent conjunctivitis. There are pain, redness, and swelling of the lids, which, however, are not tense and hard as in the diphtheritic form, and at the beginning at least can be easily everted. There is a purulent discharge after the stage of hyperæmia has passed, but it is not so abundant as in idiopathic purulent conjunctivitis. The characteristic feature is the formation of a white or grayish membrane on the surface of the conjunctiva. This may cover the whole of the surface or appear only in patches, and is usually confined to the retrotarsal folds and the palpebral surface of the conjunctiva, the bulbar conjunctiva seldom participating. This membrane can be wiped off, though sometimes only with considerable force, revealing a raw and usually a bleeding surface underneath. We often have a membrane similar to this formed on the conjunctiva as a consequence of the inflammation caused by jequirity, and even after severe cauterization with nitrate of silver or other destructive agents. It is distinctive of this form that the membrane lies on the surface and is limited to the epithelial layer, or, at least, does not go deeply into the surface of the conjunctiva. After some days the membrane begins to cast itself off, and the stage of purulency sets in. There are then the symptoms and appearances characteristic of purulent conjunctivitis. In fact, this seems to be the manner of resolution of the disease. Occasionally the membrane comes away as a whole, but usually it is detached in patches, revealing a thickened and red conjunctiva beneath, which secretes pus. It sometimes happens that there is a re-formation of the membrane and with it a return of the symptoms of hyperæmic irritation. There is not usually any systemic disturbance except a slight elevation in temperature.

The discharge is contagious, and the same precautions should be used to avoid infection of the other eye, if only one is affected, and of the eyes of the attendants, as have been laid down in the section treating of purulent conjunctivitis.

In the *treatment* of croupous conjunctivitis the same principles hold as for purulent conjunctivitis. During the hyperæmic stage irritants and nitrate of silver are to be avoided. The eyes should be kept thoroughly clean with mild antiseptics, and if there is much heat and redness of the lids cold applications are to be used. But cold is called for here even less strongly than in the early stages of purulent conjunctivitis, and in the majority of cases the use of hot water in the manner prescribed in the treatment of purulent conjunctivitis will be found to be more grateful and more efficient in hastening the casting off of the membrane. As soon as the stage of hyperæmia has passed, and the membrane begins to be detached, showing the red conjunctiva beneath, and the secretion changes from a watery flocculent

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discharge to one of pronounced purulency, then is the time to commence the use of nitrate of silver. The dosage rarely need be so strong as in purulent conjunctivitis, and it should be especially weak at the beginning, when some of the membrane still remains to be thrown off, and it is generally wise to limit its application to the parts of the conjunctiva not covered with the membrane. It is well to begin with two or three grains to the ounce of water, and, if necessary, increase the strength with the increase of the degree of purulency. Formalin would undoubtedly be useful here also, though no experience with its use has been as yet reported. The cornea is not liable to be affected as in either purulent or diphtheritic conjunctivitis, but when it is the treatment is the same as under similar conditions in purulent conjunctivitis. The conjunctiva itself is not apt, except in very severe cases, to be destroyed to such an extent as to lead to a cicatricial contraction.

Diphtheritic Conjunctivitis.—The clinical features of the diphtheritic form of conjunctivitis are not only more intense in their manifestations than those of the croupous form, but have, in addition, many distinct characters of their own. The lids are not only swollen and painful to the touch, but hard, feeling sometimes like board, and eversion of them is not possible. The conjunctiva of the lids is thickened and infiltrated with plastic material. This infiltration may extend to the conjunctiva of the ball, though commonly only in very severe cases. It is sometimes coextensive with the conjunctival surface, but usually it shows itself in areas or plaques. These areas are marked by a dirty gray, membranous patch, but as a membrane set in, rather than lying on, the surface of the conjunctiva. These gray patches cannot be wiped off, as they can be in the croupous form. When the infiltration is extensive the whole conjunctiva has a pallid look, and when incised gives out only a little bloody serum, if anything. Ecchymoses are quite commonly seen in the substance of the conjunctiva which is not already infiltrated.

The discharge at first is watery and flocculent, resembling that of a beginning purulent inflammation. It is usually only at the end of twenty-four or forty-eight hours, when the infiltration is established, with its attendant hardness of the lids, that a clear differential diagnosis is possible.

The next stage, or that of decline, is marked by an amelioration of the acute symptoms, a diminution in the hardness of the lids, and a change in the discharge to a more purely purulent character. The membrane is seldom cast off, even in patches, as it is in the croupous form, but seems to disappear by absorption; the intervening conjunctiva assumes a redder and softer appearance, and it is from this that the purulent secretion now comes. In severe cases, however, the patches slough out, leaving defects in the conjunctival substance which must heal with cicatrization. This interference with nutrition, due to the dense infiltration of the substance of the conjunctiva, is, in fact, the prime local danger of the disease. It affects not only the conjunctiva, but also, and, unfortunately, even more seriously, the cornea. It may be said, indeed, that the amount of danger can be

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gauged by the amount and extent of the infiltration into the conjunctival substance. This exudation makes pressure on the blood-vessels, diminishing the nutritive supply, and when it is at all diffused is felt particularly by the cornea, which receives its nutrition largely from the adjacent conjunctival and subconjunctival vessels. A complete sphacelation of the cornea is, therefore, no uncommon result, and ulceration of greater or less extent is seldom absent in ordinarily severe cases. It is this infiltration also which is the cause of the intense pain by the pressure it makes on the nerve-filaments.

When the lids have become soft, and there are no plaques in the conjunctiva, and the discharge has assumed the character of creamy pus, the essential diphtheritic nature of the disease may be said to have disappeared. It is no uncommon thing, however, for one or more relapses to occur, and this should be always borne in mind and its liability guarded against as far as possible by careful attention to the local and general conditions.

There is always a systemic disturbance in every marked case of diphtheritic conjunctivitis, and the depression of the vital forces is sometimes as profound as in the aggravated forms of faucial diphtheria. Frequently, in fact, there exist, concomitantly or consecutively, conjunctival, faucial, and nasal diphtheria.

The discharge is contagious: the same precautions as to infection that were given under purulent conjunctivitis are applicable here, and it should not be forgotten that diphtheritic conjunctivitis sometimes implants itself upon the muco-purulent or purulent form.

Fortunately, this disease, at least in its worst forms, is not a common one in this country, nor is it so frequent in England or France (Galezowski saw seven cases in one hundred and fifty thousand patients) as in Northern Germany. It has been known to assume an epidemic character there, particularly in Berlin, and frequently to appear as an endemic disease. It attacks by preference the young, though adults are by no means exempt, and it seems probable that the contagion can be conveyed by the air, and that its direct transference is not necessary for infection.

In the *treatment* of diphtheritic conjunctivitis depletion in the form of blood-letting is seldom called for, and certainly not after the period of infiltration has arrived. On the contrary, general sustaining and even stimulating measures are called for if the vital depression is at all marked.

In the hyperæmic stage cleanliness, with mild antiseptic or aseptic solutions and cocaine, is indicated, with anodynes internally if the degree of pain calls for them, as it very frequently does. These same measures are to be continued during the stage of infiltration. In carrying them out, however, and particularly the cleansing, we are very much hampered by the fact that the lids are so hard and unyielding as to make eversion and sometimes even separation impossible, and it should not be attempted in any forcible manner, on account of the great pain attending the effort. The patients should be put to bed and treated as though suffering with a serious

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ailment. The period of usefulness of cold applications is shorter here even than in the purulent and croupous forms, and is limited entirely to the stage of hyperæmia. When the stage of infiltration is thoroughly established, the application of cold, especially if continuous, is likely to diminish still further the vitality of the parts, already much impaired, while, on the other hand, the application of heat has an alleviating effect on the pain and tends to hasten the process of resolution.

The application of hot water in the manner described under purulent conjunctivitis should, therefore, be employed much more frequently and for a longer time than in either that or the croupous form; for the danger of destructive ulceration from impaired nutrition is greater, so far as the conjunctiva itself is concerned, than in purulent conjunctivitis. It is not advisable, unless the indications urgently demand it, to make a canthotomy for the relief of pressure, for the diphtheritic inflammation is liable to attack the wound and thus add to the complications of the disease.

The period of infiltration may last from two days to six or more, according to the intensity of the attack, and the signs of its beginning subsidence are a change in the character of the discharge, which becomes more purulent, and a softening of the lids. When these become manifest we may assume that resolution has set in. The conduct of the case during purulency or resolution does not differ in principle from that of purulent conjunctivitis itself, though it requires much tact and judgment in carrying it out. For purulency, of course, nitrate of silver is the remedy, but it must be used with caution at the beginning, for its application in strong solutions is known to have been followed by an exacerbation of the symptoms and a re-formation of the membrane. From some experiences reported with formalin in faucial diphtheria, this drug would seem to be a remedy likely to act well in this stage. Solutions should vary in strength from 1 to 2000 to 1 to 500, according to the degree of purulency. As the resolution, like the infiltration itself, usually takes place in areas and not throughout the conjunctiva as a whole, there are, even during the period of purulency, patches of exudation which have not undergone resolution. It is therefore wise in making the applications of the silver nitrate to limit them to the parts which are vascularized and free from infiltration and which are giving out the pus, and to wash off carefully the superabundant salt. Neither must the solution be strong. It should not exceed from one-half to one per cent. at the commencement of the treatment, to be increased later if the degree of purulency demands it.

Many topical remedies have from time to time been suggested and tried to assist in the resolution of the exudation, such as citric acid, powdered quinine sulphate, etc., but none of them have come to anything like a general acceptance in the therapeutics of the disease. Some good authorities recommend the administration of mercury internally, with the view of hastening the absorption of the exudation and preventing its recurrence. For this purpose from five to ten centigrammes of calomel are to be given every

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two hours until the effects are manifested. This may be assisted by inunctions of mercurial ointment.

When the cornea has become ulcerated, it should be treated by atropine and hot applications. Cauterization of the ulcers should not usually be attempted in the first stages, since they are due, for the most part, to lowered vitality and not altogether to microbic infection. Later, however, when resolution is setting in and vitality has returned, cauterization with the actual cautery or with formalin 1 to 60 is useful.

During the stage of cicatrization care should be taken to prevent adhesion between the lids and the ball, which is likely to take place when the two raw surfaces are brought together. The lids should be moved frequently over the surface of the ball, and it may be necessary to introduce a bit of thin charpie between them to keep the raw surfaces apart. Where this is not thought advisable, the free use of vaseline is recommended for the same purpose and as a protection of the raw surfaces.

While this article is being written, the value of the *serum-therapy* in diphtheria is under discussion, with a large preponderance of opinion in favor of its usefulness. Its employment, therefore, will probably form a prominent feature in the future treatment of those forms of conjunctivitis in which the Klebs-Loeffler bacillus is found; in fact, many cases successfully treated in this way have been already reported.

The entropion which results from the cicatricial contraction of the conjunctiva is to be treated surgically in the same way as that arising from trachoma or other cicatricial shrinking of the conjunctiva.

A *chronic form* of membranous conjunctivitis has been observed by Nettleship (1880), Critchett and Juler (1883), Howe (1897), and others, in which the membrane is formed and thrown off almost daily for weeks at a time, the whole disease lasting many months. The membrane is on and not in the conjunctiva, and the conjunctival substance is not destroyed. In Howe's case a streptococcus was the microbe found.

SCROFULOUS, LYMPHATIC, OR STRUMOUS CONJUNCTIVITIS (PHLYCTENULAR CONJUNCTIVITIS; HERPES CONJUNCTIVÆ).

The forms of conjunctivitis we have hitherto considered are to be classed among the infectious diseases, so far as they depend primarily upon the introduction of pathological germs from without, though it presupposes a ground prepared for their growth and development. There is another quite distinct class of conjunctivitides which are to be regarded as expressions of dyscrasiæ, and though they may and probably do require a special germ for their causation, yet the condition of the general system and the state of the nutrition are important if not the prime factors.

The dyscrasia known as scrofula or struma manifests itself in the conjunctiva more frequently than any other disease of malnutrition, and is especially frequent in children, though adults are by no means free from it. It must not be understood, however, that the appearance of vesicles on

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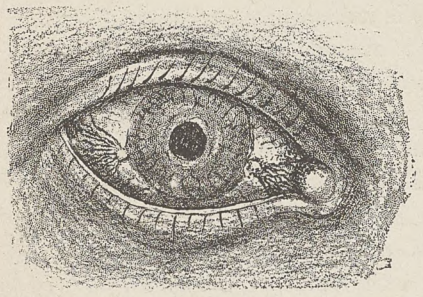
the conjunctiva is a certain indication of established scrofula, for we often find them during a temporary lowering of the nutritive powers in children who are apparently strong and show no evidences of the dyscrasia elsewhere, and there are cases of pronounced and severe forms of strumous disease in which the conjunctiva is slightly or not at all affected. Still, the appearance of these vesicles on the conjunctiva can be accepted as a certain sign of faulty assimilation, and generally of an impoverished condition of the blood.

FIG. 2.



Scrofulous conjunctivitis; phlyctenulæ,—one on each side of the cornea.

It would seem probable, too, that in some instances at least the vesicles are the manifestations of a derangement at the nerve-centres analogous to, if not identical with, that causing herpes zoster. We can hardly account otherwise for the very pronounced nervous symptoms, such as severe pain, lacrymation, and photophobia, which we not uncommonly see. There is no other disease which varies so widely in the intensity of its symptoms; and, what is still more remarkable, the subjective symptoms do not seem to bear any direct relation to the objective manifestations. Frequently a minute vesicle, especially if situated on the cornea or on the sclero-corneal margin, will be associated with the most intense pain, photophobia and lacrymation persisting for days and weeks, while two or three large ones scattered over the conjunctiva give rise to comparatively little inconvenience. This is due, probably, to two causes, separately or combined,—namely, the extreme susceptibility of the nervous system and the pressure made directly on the filament of a nerve by the exudation. This latter is more apt to be the case where the vesicle is situated on the conjunctival layer of the cornea, where the nervous supply of the fifth pair is more generous, and the arrangement of the terminal filaments among the epithelial layers is such as to lead more readily to such pressure. In the same way we may account for differences in the severity of various attacks in the same person, one being mild, and another, with the same objective appearances, very severe.

The location of the exudate may be on the conjunctiva proper, or in the epithelial layer of the cornea, which is continuous and anatomically identical with that of the conjunctiva of the sclera. The exudate lies between the epithelium and the layer of Bowman, and is not, as a rule, fluid, but consists of an aggregation of round lymphoid cells, as shown in Fig. 3. The disappearance of the exudate takes place from the apex in the form of an ulcer, which, however, soon heals over by the formation of the new epithelium and leaves no scar. When the process extends deeper, which it sometimes does, and affects the layers of the cornea proper, causing a genuine

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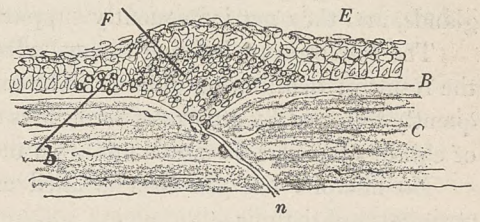
keratitis, the exudate may still be absorbed without a scar if there has been no loss of the corneal tissue. But if the corneal tissue has been destroyed its place is filled with cicatricial tissue which remains opaque. These ulcers on the cornea not infrequently assume a serpiginous character, creeping over the surface and being followed by a leash of vessels, the so-called *fascicular keratitis*. These manifestations, however, are due to the implantation of another infection on the ulcer caused by the phlyctenula.

The characteristic *pathological change* is what appears to be a vesicle which varies in size from that of a pin-head or even less to a bleb three or four millimetres in diameter and approaching that of a bulla.

FIG. 3.



Phlyctenula conjunctivæ.—*C*, cornea; *B*, Bowman's membrane; *E*, epithelium; *F*, collection of lymphoid cells under the epithelium; *b*, lymphoid cells among the epithelial cells; *n*, nerve filament with lymphoid cells along its course. (After Iwanoff.)

The exudate is just under or in the epithelial layer, and the contents are usually clear and seldom purulent; it is not fluid, but, as has been noted above, consists of a collection of lymphoid cells. The associated conjunctival vascularization may be only a bunch of fine vessels running from the equator of the ball forward to unite at the base of the phlyctenula (Fig. 2), the remainder of the conjunctiva being perfectly clear, or it may be general and diffuse, approaching that of a muco-purulent conjunctivitis. In all but exceptional cases, however, the conjunctival changes are most pronounced around the phlyctenula. Though usually single, there may be any number of these phlyctenulæ, and sometimes they form a circlet around the base of the cornea. There is frequently associated with the conjunctival disease a swollen condition of the edges of the lids, and sometimes of the whole lid, due to a concomitant affection of the tarsus. The discharge is watery in the cases where there is a great deal of photophobia or where the other nervous symptoms are prominent, but where there is much associated general conjunctivitis it is mucous or muco-purulent. The discharge from the eye is often very acrid and causes an eczema of the cheeks over which it flows and of the lips and the inside of the nose. In badly nourished and ill-cared-for children the whole face is sometimes covered with scabs and excoriations as a result of this irritation. In cases of persistent blepharospasm we not infrequently find excoriation and ulceration in the folds of the skin at the outer canthus, which, by reflex action, tend to keep up the spasm of the orbicularis.

When the blepharospasm is very intense and obstinate, with considerable swelling of the lids and tarsus, there is sometimes a total eversion of the lids (ectopia conjunctivæ), which may remain even after the original cause has subsided. The actual pain, outside of the photophobia, is not usually great. The dread of light, however, which is sometimes found in young

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children is extreme. They seek the darkest corner of the room and bury the whole face deep down in a pillow, the least ray of light seeming sufficient to cause them the greatest agony even when falling on the tightly closed lids. This by keeping the patient closely housed and by preventing proper exercise tends greatly to depress still lower the vital forces and thus aggravate the fundamental cause of the disease.

The appetite is nearly always poor, and very frequently depraved, there being a craving for indigestible and unwholesome articles of food. Candies, sweetmeats, and pastry are preferred to more nutritious substances. There is nearly always a swelling of the submaxillary and the preauricular glands, and they not infrequently suppurate.

The condition underlying the eye affection may be idiopathic or may be the result of debilitating disease. The exanthemata are, perhaps, more frequently followed by attacks of scrofulous ophthalmia than the other diseases of childhood. The disease is not often met with under the first year of life.

No microbe has yet been isolated as the special agent in producing strumous ophthalmia.

In the *treatment* of scrofulous conjunctivitis we are governed largely by the severity of the attack. In the mild cases, where there is no photophobia and but little attendant conjunctivitis, the local treatment should be limited to a mild antiseptic, as boric acid solution, or sodium biborate solution, gr. x ad ℥i, dropped in the eye three times a day, with protection of the eyes from overwork and from the bright glare of sunlight and artificial light by shades or protective spectacles, with perhaps a tonic of quinine and iron, and attention to diet and sleep. Plenty of out-door exercise should be insisted on even in cases where the photophobia is considerable. In such cases the exudation usually disappears in from four days to a week, leaving no ill effects behind.

The severest cases, however, tax the skill and patience of surgeon and attendant alike to the utmost. Every attempt to make an examination or to do anything to the eye is a struggle, and yet this must be done two or three times a day. In the less severe cases, where the photophobia is not great, a little quiet coaxing will often succeed in obtaining for us a view of the cornea. Place the little patient with its back to the light, and, without touching the eye, try to get it to follow the finger or a watch or other object held in the hand as it is moved in front of it. Nearly always this succeeds in obtaining some separation of the lids. The moment the eye is touched, however, it will be tightly closed, and force is then usually necessary to open it.

The first examination, which should be thorough, it is best to make under chloroform when the photophobia is very intense or the coaxing method fails. It seems less inhuman, and there is less danger of doing injury to the eye than in the effort to open the lids forcibly. This latter, however, must be the usual proceeding, and it should be performed with exceeding care. The head of the child should be held firmly between the

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knees of the operator while its arms and legs are restrained by an attendant. The lids being wiped dry, the forefinger of the right hand is placed on the edge of the upper lid and the whole lid pushed firmly backward and upward, while the thumb of the left hand pulls the lower lid downward. The eyeball is then uncovered to examination. Care must be taken to hold the edges of the lids firmly against the ball, otherwise the lid will be everted and the cornea covered by the exposed tarsus. The local applications in severe cases should be soothing, and among the remedies cocaine and atropine are the best. A drop of a one per cent. solution of each should be put in the eye three times a day. They can be used singly or combined. Cocaine has a good effect on the photophobia and blepharospasm, and if employed first makes the use of atropine easier and more effective.

The excoriation of the face caused by the acrid discharge should be treated by oxide of zinc ointment or by an ointment of the yellow oxide of mercury, gr. i to ʒii of cosmoline. The fissures of the outer canthus, if deep, should be thoroughly cauterized with nitrate of silver. Their healing is often accompanied by a prompt amelioration of the blepharospasm. Another most efficient remedy for blepharospasm is the immersion of the face of the child in cold water. It usually struggles violently against this, but when its head is taken out of the basin, in its effort to regain breath it frequently opens the lids wide which have been closed tightly for many weeks. Another method is to keep the lids forcibly apart with a spring speculum for some minutes once or twice a day.

If the ulceration has extended below the epithelium into the tissues of the cornea proper, and particularly if it shows a tendency to spread, cauterization of the ulcer is valuable both in stopping the progress of the ulcer and in allaying the photophobia. This may be done by the cautery (actual or galvano-), nitrate of silver, or carbolic acid. This latter I have found very satisfactory when applied after the ulcer had been scraped clean. Formalin 1 to 60 can be used for the same purpose. In those cases where the conjunctivitis is pronounced and there is a mucous or muco-purulent discharge, and where photophobia is no longer present in any considerable amount, nitrate of silver two or three grains to the ounce, applied to the conjunctiva twice a day, is indicated, as well as the other treatment of this form of conjunctivitis.

From the earliest times, almost, calomel has been used as a local application in this form of conjunctivitis. A quantity of calomel powdered as finely as can be is dusted directly into the eyes once or twice a day. It is probably slowly converted into the bichloride and thus operates as an antiseptic. It should not be used at the same time that the iodides are given internally, since the two will form a very caustic mercuric iodide which is highly irritating. With the same therapeutic object in view, the salve of Pagenstecher (hyd. oxid. flav., gr. ii, cosmoline, ʒii) may be introduced into the eye and afterwards distributed over the surface by gently rubbing the lids. Attention to the general condition of the patient is of prime importance.

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Everything should be done to improve the nutrition and assist the assimilation. Only the most nutritious articles of food should be allowed, and cod-liver oil is always beneficial. The articles of diet most strongly indicated are milk, eggs, beef, mutton, and fowl; no pork or veal; very little bread or potatoes; no sugar or fat; tea and coffee in great moderation. Food should be taken frequently rather than in large quantities at a time. Of drugs, the iodide of iron is that which is most generally used. From fifteen to thirty drops of the syrup in half a glass of water should be given to a child three times a day. The patient should be in the open air as much as possible, and should be taken in summer to the mountains rather than to the sea-shore, on account of the glare of the latter. Bathing in salt water, however, is a valuable therapeutic agent.

If there be any concomitant disease of the nasal passages, it should be treated thoroughly. There can be no question that, in children especially, there is a close connection between the nasal and the conjunctival inflammations, and the treatment of the nose-trouble is promptly followed by an amelioration or cure of the conjunctival inflammation.

School-children in particular should be carefully examined as to their optical condition, and all anomalies of refraction and disturbed muscular balance carefully attended to.

GRANULATION OF THE CONJUNCTIVA (GRANULAR LIDS).

Under this heading we shall consider those diseases of the conjunctiva whose chief characteristic is a granular or uneven appearance of the conjunctival surface.

Concerning these diseases the most widely differing opinions are held as regards nature, causes, pathology, and treatment, and a full exposition of these varying views would require a volume to itself. They are thought by some to be purely local affections, by others to be the expressions of some generally operating cause. They are regarded in some quarters as highly contagious, in others as hardly contagious at all. Some claim to have found their specific microbes, while other equally competent observers deny their demonstration. The methods of treatment in detail are as numerous as the authors who have written about them, or almost as the individual practitioners. The literature on the subject is immense in its mass, and a study of it is apt to lead to confusion rather than to clarity of opinion concerning one of the most serious diseases to which the eye is liable. The most that can be done, therefore, within the limits allowed to this article is to express the personal opinion of the writer on these various points, based on his observation and reading, without reciting a history in detail of the numerous theories and practices that have been advanced from time to time by investigators and students in various parts of the world, each of which has probably contained some grain of truth that will at last find an abiding-place in the golden granary of scientific knowledge.

"Granular lids" is not, from a clinical point of view, a single disease.

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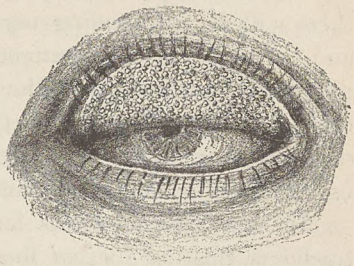
It may be that the pathologist of the future will be able to demonstrate a common origin of the different varieties of granular lids met with clinically, but with our present knowledge this cannot be confidently affirmed, and we are justified, by their course, pathological changes, and termination, in making at least two broad general divisions which can very well be studied independently as regards their clinical manifestations and treatment. These are simple granular or follicular conjunctivitis and trachoma.

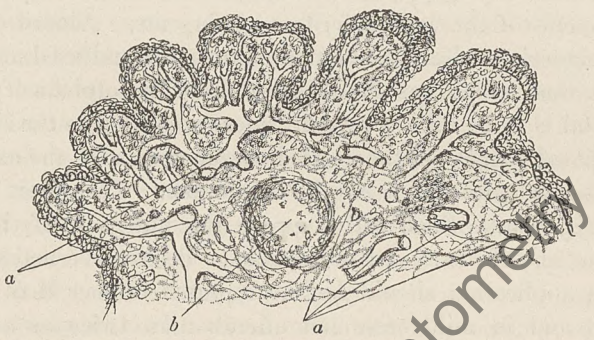
FIG. 4.



Follicular conjunctivitis with no thickening of the conjunctiva.

Simple Granular Conjunctivitis (Follicular Conjunctivitis).—In this form of disease the surface of the palpebral conjunctiva (Fig. 4) is covered over with granules, varying in size from that of a rape-seed to the point of a pin, evenly distributed, and usually in rows parallel to the edge of the lid. They are reddish or yellowish in color, and

FIG. 5.



Follicular conjunctivitis, showing the enlarged papillæ with their vascular supply, *a*, *a*. (After Raehlmann.)

the conjunctiva beneath may be thickened, but is soft and pliable. They are simply the enlarged or hypertrophied normal papillæ or follicles of the membrane (Fig. 5). They are sometimes the result of a long-continued, though it may be low and mild, form of inflammation or simple congestion of the parts. The condition is also called *hypertrophied papillæ*. It frequently remains as a sequela of purulent or muco-purulent conjunctivitis, but often the beginning is difficult or impossible to fix. Stephenson (1895) considers it in its simplest form as the manifestation of an adenoid activity, and to that extent physiological. It was present in seventy-five per cent. of school-children examined by him. It has this distinction, however, that when pathological the hypertrophy is due to an ordinary inflammatory exudation and enlargement of the normal papillæ, with no tendency to a destructive process however long the inflammation may be continued. It never runs into trachoma. The accompanying symptoms are those of chronic

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congestion or of catarrhal or purulent conjunctivitis, according as the one or the other process in the conjunctiva is the more active. There is a feeling of discomfort about the eyes, heaviness of the lids, more or less inability to use the eyes, particularly by artificial light, and if the secretion is active there will be a gumming together of the lids on waking in the morning and formation of crusts among the lashes. The cornea is not necessarily nor usually involved, and the bulbar conjunctiva may remain clear except during an exacerbation due to some acute inflammatory condition or an exposure to irritating influences, such as dust, or smoke, or long-protracted use of the eyes under bad illumination, or to loss of sleep.

In some instances this form of granulation may be grafted on true trachoma, making a *mixed* form. Indeed, in most cases the irritation produced by the trachoma deposit gives rise to a more or less hypertrophic state of the normal papillæ of the conjunctiva. Follicular conjunctivitis has this distinctive feature, however, as we have said, that however long it may continue it does not lead to a destruction of the conjunctiva nor to an inversion of the lids.

The *therapeutic indication* is to reduce the hypertrophy by an absorption of the inflammatory product. This can be best accomplished by the local use of some of the various irritant astringents. Almost every astringent in the materia medica has been recommended and used at one time or another, but that which has found most favor is sulphate of copper. A smooth crystal is to be rubbed several times (according to the effect desired) over the exposed surface of the everted conjunctiva, and the excess washed off with water by means of a camel's-hair brush. The object is to cause a temporary hyperæmia which will improve the nutrition of the parts and stimulate the absorbents. An escharotic or destructive action is not desired. This application should not be repeated oftener than every other day usually, and in mild cases not oftener than twice or once a week. During the interval zinc. sulph. gr. ii ad ℥i should be used as a collyrium twice a day, and in very mild cases where the hypertrophy is not pronounced this may itself suffice without the use of the copper crystal. A stick of alum can be used for the same purpose, but its action is milder. Should there be any considerable secretion, a one per cent. solution of nitrate of silver may be applied, but only for a limited time, on account of the staining of the conjunctiva which follows its prolonged employment. Glycerin and tannin (gr. x ad ℥i) and boroglyceride have been used with good effect, applied once a day. Acetate of lead is useful. The care of the eyes is a most important element in treatment. Use by artificial light is nearly always harmful, and should be avoided, as also should dust and smoke and vitiated air of any kind.

In every case the condition of the nasal mucous membrane should be examined and any fault found there removed. Any existing ametropia should be carefully corrected. Properly fitted glasses sometimes relieve the milder cases as if by magic.

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Even in those cases in which a suspicion of the coexistence of the trachomatous deposit is not strong, but where there is considerable swelling of the papillæ and the condition has become chronic, the mechanical treatment by "squeezing," with previous slight scarification, will, in my opinion, be of benefit in unloading the tissues of inflammatory material which would be absorbed only with difficulty and after a long time.

Trachoma.—Of all the inflammatory diseases of the conjunctiva this is of most importance, on account of its wide-spread diffusion, the chronicity of its course, and the disastrous results which are liable to attend it. Purulent conjunctivitis may be more immediately destructive, but fortunately it is not of such frequent occurrence. In some countries, as Poland and Russia, trachoma is the cause of almost fifty per cent. of all the blindness, and forms from thirty to ninety per cent. of all eye diseases presenting for treatment.

It has been supposed that the disease was introduced into Europe in 1802 by the soldiers of Napoleon returning from Egypt, where a contagious ophthalmia constantly abounds. It is very doubtful, however, whether all the inflammatory affections of the conjunctiva which were observed at that epoch were true trachoma as we at present recognize it. It is certain that Egyptian ophthalmia, as we now know it, includes several distinct forms of conjunctivitis, and that trachoma is only one of them. Its origin undoubtedly dates far back into antiquity.[1] Myjashita says it was known in Japan more than twelve hundred years ago.

It is difficult to classify trachoma otherwise than clinically. No microbe has yet been discovered which all investigators unite in acknowledging as peculiar to it,[2] and the pathological alterations found in the early stages are not sufficiently distinctive or constant to warrant a differential diagnosis on that ground alone. But the clinical picture in advanced stages is quite clear and unmistakable, and makes an error in diagnosis impossible.

Trachoma is a disease of the conjunctiva, mostly chronic in its course, though subject to exacerbations and remissions of inflammation, due to a morbific deposit or change in the tissue, which in time causes a destruction of the parts surrounding it, a cicatrix being left behind as the result of its disappearance, with a marked tendency to contraction.

The appearances and manifestations must, therefore, differ widely in the different stages of the disease, and this circumstance has given rise, among other causes, to a classification of trachoma into numerous varieties, such as "acute," "inflammatory," "mixed," etc., each of which is only a particular and special phase of one affection. The manifestations which are usually most prominent are those due to the inflammation of the conjunc-

[1] For an account of granular conjunctivitis among the ancient Egyptians, Greeks, and Romans, the reader is referred to an article by Dr. J. Hirschberg, contained in Chibret's "Etude de Géographie ophthalmologique," Paris, G. Steinheil, 1896.

[2] See article on "Micro-Organisms of the Conjunctiva," by McFarland and Kneass, in vol. iii. of this System.

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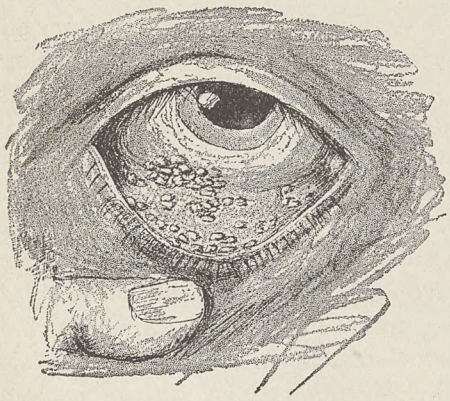
tiva. This may be any one of the forms already treated of, namely, hyperæmic, congestive, catarrhal, or purulent, and the appearance of the conjunctiva will be that distinctive of the particular form present, superadded to that which is characteristic of the disease itself, namely, a peculiar "granular" look of the conjunctiva of the lids, which has given the popular name to the disease. In most cases of true trachoma in a somewhat advanced stage these "granulations" are merely the greatly enlarged normal papillæ, the hypertrophy being induced by the long-continued inflammation, and they often hide the true trachoma granules from view.

The genuine trachoma granule is seen best in the early stages of the disease and before it has brought about the inflammatory changes above mentioned. It appears then as a small, round, grayish, opaque granule, from one to two millimetres in diameter, embedded in, but rising above, the level of the conjunctival surface (Fig. 6). These granules are confined almost wholly to the palpebral portion of the conjunctiva, particularly that of the upper lid and the retrotarsal folds. On the conjunctiva of the ball they are rarely seen. They may be limited to a small portion of the surface of the membrane, or may occupy its entire extent, and are irregularly placed, often in masses. They look not unlike the spawn of frogs or small grains of sago, and have been called "sago-grain granulations" or "frog-spawn granulations." They are said to have been frequently found in the conjunctivæ of eyes which have never shown any signs of inflammation, and which have been regarded as healthy (Nettleship and others); though some, as Stephenson, regard these as only enlarged normal follicles, and claim that they never become trachomatous. From whatever source they may come and howsoever they may be formed, these are the essential pathologic elements of the disease, and they are at the foundation of all the changes and varied phenomena which form the clinical features of the affection. It would seem that they act as foreign bodies very much in the same way as tubercles do, and, like tubercle, lead to inflammation and destruction of the tissue in which they are embedded.

FIG. 6.



Trachoma granules in the early stages. (From a photograph after Cohn.)

The following table, slightly modified from Stephenson ("Epidemic Ophthalmia," 1895), gives the differential diagnosis, point by point, between folliculosis of the conjunctiva and trachoma, as accepted by the majority of authorities at the time of this publication:

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False or Follicular Granulation.	*Trachoma.*
1. Oval or roundish transparent bodies the diameter of which never exceeds from one millimetre to one and a half millimetres. Of a faint yellowish hue, arranged in rows parallel to the lid border, and discrete. Most marked in inferior retrotarsal fold.	1. Round, opaque, ill-defined bodies, of grayish-white color and extreme friability. Firmly and deeply embedded in the conjunctiva, their diameter not infrequently reaches two millimetres or more. Tendency to become confluent and form masses or areas of trachomatous material. Most numerous and larger in upper retrotarsal fold.
2. Little or no change in the structure of the conjunctiva.	2. Structural changes always present.
3. Papillary hypertrophy of upper lid slight.	3. Marked hypertrophied papillæ of upper lid generally present.
4. Tarsus never implicated.	4. Tarsus often involved.
5. Disappear spontaneously generally and leave no scar.	5. Spontaneous cure may occur, but only by cicatrization, which may be slight or extensive according to the amount of tissue involved.
6. No ptosis.	6. Ptosis nearly always present in some degree.
7. No pannus.	7. Keratitis in the form of pannus or ulcer in about twenty-five per cent. of the cases.
8. No trichiasis, entropion, or cicatricial contraction of the cul-de-sac.	8. Frequently leads to trichiasis, entropion, or shrinking of the cul-de-sac.
9. Most frequent in persons under twenty years.	9. May occur at any age.
10. Non-contagious.	10. Conditionally contagious.

As we seldom see these cases until the inflammatory symptoms send the patient to the surgeon, there has always been a question as to whether the deposit is the cause or the result of the inflammation. From the fact above stated, however, that they have been found in eyes which have not been inflamed, it would appear more likely that the inflammation is not the first step in the process, though undoubtedly the inflammation, when it is once set up, facilitates its progress and encourages new deposits, and thus a vicious circle is completed.

It is this which has given rise to the mistake of classifying all the ophthalmias of the East under the head of trachoma. The conditions of life there are such as to lead to the production of severe forms of conjunctivitis, and all forms of conjunctivitis are favorable to the development of trachoma wherever there is a natural tendency in that direction.

The *symptoms* of trachoma in the inflammatory stage uncomplicated with corneal troubles are those of conjunctivitis, and usually of the mucopurulent type, less commonly of the simple congestive or catarrhal form. The lids are usually thickened, and there is a partial ptosis. The inside of the lids, when they are everted, is seen to be roughened or "granulated," and sometimes these granulations are enormous in size. In the stage of cicatrization the inside of the upper lid particularly, which seems to bear

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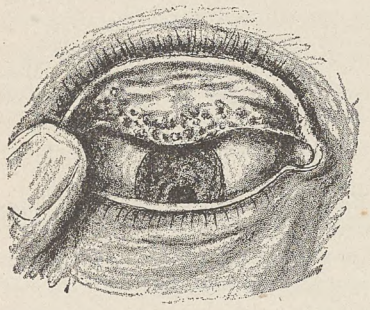
the brunt of the disease, assumes a hard, gristly appearance, and there are usually lines of cicatrization running lengthwise of the lid, as shown in Fig. 7. This destruction of the conjunctiva with formation of cicatricial bands varies from a line of white faintly seen in the swollen conjunctiva to a surface of yellow, dry, and rough cartilaginous appearance without a tinge of redness.

FIG. 7.



Secondary stage of trachoma, showing cicatrices of the conjunctiva.

There are the usual discomfort and inability or difficulty in using the eyes, especially by artificial light, the feeling as of a foreign body in the eye, and the sensations of burning, itching, and heaviness of the lids, etc., which are the common accompaniments of the various forms of conjunctivitis. Occasionally, however, the subjective symptoms are almost *nil*, and the disease is manifest only on an examination of the lids. When the cornea becomes implicated, as it almost always does at some time during the course of the disease, we have a totally different set of phenomena. There are then all the signs of corneal inflammation, such as increased lacrymation, photophobia, and pain of a neuralgic character, all of which may vary in degree from the slightest possible to the most severe. This corneal complication may occur in the earlier periods of the disease, but it usually appears in any considerable intensity only when the stage of cicatrization of the conjunctiva has set in. The keratitis is to a large extent a purely mechanical one, due to constant friction of the rough conjunctiva over the surface of the cornea, or to the rubbing of the inturned cilia over the ball. This form of keratitis is known as *pannus*, and has been subdivided into two forms, *pannus crassus* when it is thick, and *pannus tenuis* when it is slight. In the first form the surface of the cornea is so overlaid with newly formed vessels and tissue as to appear like a fleshy mass which hides the iris totally from view. It is asserted that the trachoma granule may appear on the surface or in the tissue of the cornea itself, and that the pannus is but the manifestation of the trachomatous process on the cornea, since the corneal trouble often shows itself before the inside of the lid becomes rough. There are many facts which point to the probability of this being true, especially when the pannus occurs very early in the disease and is limited to the epithelial layer of the cornea.

One of the most serious complications or results of trachoma is the incurving of the tarsus caused by the contraction of the cicatrix following the destruction of the conjunctiva and of the adjacent part of the tarsus itself. This condition is known as *entropion*. (See article on Diseases of the Lids.)

The incurving of the tarsus when it is of any considerable degree brings

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the eyelashes in contact with the ball, and these by their constant irritation keep up the keratitis, and, in fact, constitute one of the most important agents in the majority of cases in bringing about the disastrous results of trachoma. For we must bear in mind here, as in blennorrhœa of the conjunctiva, that it is the integrity of the cornea which is endangered and which we have to guard. The inflammation of the cornea is not always of the proliferative form (pannus) of which we have spoken. The keratitis often ends in ulceration or softening of the tissues, resulting in partial or total opacity (leucoma) or staphyloma, or even, in very severe cases, in panophthalmitis and atrophy of the ball.

As has been already mentioned, one characteristic of the disease is its proneness to exacerbations and remissions. This may be accounted for, partly at least, on the assumption of a fresh deposit of the trachomatous material at each renewal of the acute inflammation. The attacks can often be referred to an exposure to unusual irritating influences, such as smoke, dust, vitiated atmosphere, or sudden change of temperature. It can continue with periodical attacks, though it may be at long intervals, from childhood to old age.

Very young children are not so likely to be attacked, probably because the adenoid tissue of the conjunctiva is not yet fully developed, and its ravages are greatest in adolescence and the early years of maturity. The male sex is somewhat more largely affected than the female. It is liable to become what is considered epidemic, especially among the inmates of boarding-schools, asylums, barracks, etc., where a number of people are collected together, and particularly where the hygienic surroundings are not good. This should not be regarded, however, as an evidence of an epidemic in the proper sense of the term, since an epidemic implies the presence of a microbe, and for trachoma its positive existence has not yet been demonstrated. The most probable explanation of these numerous cases at the same time and place is that the conditions are from some cause or set of causes such as to lead to the development of the disease in those in whom there is a latent tendency to the trachoma deposit.

It is held by many that the disease is eminently contagious, but evidence is lacking to prove that such is the case in the acceptance of the word as we understand it in regard to ophthalmia purulenta, for instance. One member of a family may be affected with trachoma for years without communicating it to any of the others with whom there is a daily intimate association, and there may be trachoma in one eye without the other ever becoming affected. Gunning, of Amsterdam, examined a school in which there was a large number of trachomatous subjects. A year later there was only one additional case. It must be understood, however, that he recognizes true trachoma only under the chronic form. Vennemann (1889) also reports against the contagiousness of true trachoma, and a number of other accurate observers have recently seen reason seriously to question it. The fact of several cases occurring in the same house is not a proof of the con-

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tagiousness of trachoma. If it proves anything, it is that the discharge from a trachomatous eye may have the germs of a purulent infection in it, and these may arouse a latent trachoma tendency in an eye into which they find entrance. It is very questionable whether the acute ophthalmia so often referred to as breaking out in the slave-ship Rodeur, in 1819, was true trachoma as we now recognize it. It appears to have been an acute purulent conjunctivitis of a virulently contagious type. It was the custom at that period to call all serious inflammatory affections of the eye trachoma. Thus far inoculation of the trachomatous material has not been successfully accomplished in a sufficient number of cases to demonstrate its infectious character beyond dispute. Van Millingen found that the introduction of trachomatous tissue into an eye did not produce trachoma, but that the secretion of a trachomatous eye sometimes did. The purulent discharge which is associated with trachoma, and which is one of its manifest symptoms in what may be called the acute stage, can, without doubt, give rise to an inflammation of a purulent type in the conjunctiva of an unaffected eye, and, if there is a predisposition to trachoma, may in this way cause a development of the disease, just as an attack of pneumonia is liable to lead to an outbreak of tuberculosis in the lungs of one who is predisposed to it. Muttermilch holds that any inflammation may lead to trachoma, but that trachoma is not contagious *per se*. The vast majority of patients affected with trachoma are among the very poor and those living under bad hygienic conditions. It has been called a disease of filth, poverty, and overcrowding, and certainly these conditions hasten a development of the disease in those predisposed to it.

It would seem, then, taking into consideration all these facts and others yet to be mentioned, that trachoma is not a simple local disease, due directly to a specific infection by a special germ from the outside, but is the local manifestation of a dyscrasia. This opinion I announced as far back as 1876 in a paper read before the International Ophthalmological Congress in New York, and the consensus of opinion is tending more and more that way. In its general course, behavior, and results it bears a close resemblance to tuberculosis (first noticed by Fallot in 1838), without being identical with that affection. Both are deposits or developments of foreign material in the tissue which lead to its destruction and the formation of contracting cicatrices; both are liable to successive depositions, and both require for their proper development what is known clinically as "predisposition," or a constitutional susceptibility to the peculiar exciting cause. And yet those who are predisposed to the one affection are not necessarily or, in fact, commonly liable to the other.

Race and country undoubtedly play an important *rôle* in the causation and development of trachoma.

Krüdener, in a flying visit (1895) through the provinces of Russia to attend the poor affected with eye diseases, among 2800 patients found 1416 affected with trachoma and its results. Cohn reports in Breslau, in 40,000

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eye patients, 9 per cent. of trachoma. Mooren, in Düsseldorf, in 127,648 eye patients, noted 7 per cent.; Schmidt-Rimpler, in Marburg, 12 per cent.; Saemisch, in Bonn, 16 per cent.; Burow, in Königsberg, 27 per cent. In Dorpat, Russia, it was 18 per cent.; in Riga, 14 per cent.; in St. Petersburg, 7 per cent. Among the Jews in Russia it is wide-spread. In Jassy, Crainician had 52 per cent. of trachoma in 2176 patients. In Rosmini's clinic in Milan trachoma ranged from 58 to 67 per cent. of all the eye cases. Van Millingen, in Constantinople, found in 5917 eye patients (cosmopolitan) 18.3 per cent. trachomatous. In some tables published by Van Millingen in the *Annales d'Oculistique*, September, 1895, we find that Holland has 7.05 per cent.; Norway, Sweden, Denmark, Switzerland, practically none; Scotland, 0.7 per cent.; England, 0.07 per cent.; Ireland, 3 per cent.; France, 4 per cent.; Belgium, 4 per cent.; Hungary, 12 per cent.; Bulgaria, 20 per cent.; Greece, 25 per cent.; Italy, 25 per cent.; Portugal, 25 per cent.; Spain, 11.09 per cent. These figures are only approximative, and many of them will, no doubt, be greatly modified by further statistics. It is common in China and Japan, and affects the Chinese and Japanese residing in this country. For England, Scotland, and Ireland, the following part of a table from Stephenson[1] is more definite:

ENGLAND.

Place.	Observer.	Period.	Total Number of Cases.	Trachoma.	Per Cent.
Manchester	A. Hill Griffith.	12 years.	192,296	1624	0.84
Bath	Beaumont.	7 years.	10,000	73	0.73
Maidstone	P. T. Adams.	30 years.	74,000	733	0.98
Birmingham	Priestley Smith.	10 years.	19,346	98	0.5

SCOTLAND.

Edinburgh	G. A. Berry.	. . . .	46,000	305	0.65
Glasgow	F. Fergus.	44 years.	222,636	1586	0.712
Dundee[1]	McGillivray.	1 year.	3,983	58	1.45

[1] Dundee has a large Irish and Jewish population, which accounts for the unusually large percentage of trachoma there.

IRELAND.

Dublin	Swanzy and Benson.	13 years.	64,223	2494	3.88
Belfast	J. Nelson.	9 years.	5,136	119	2.31
Belfast Children's Hospital	J. Nelson.	8 years.	2,618	32	1.22

These statistics show that the frequency of trachoma varies in different countries from 0 to 67 per cent. or even more. We are well aware, in a general way, of the great unreliability of statistics and how they can be made to substantiate almost any position, yet so great a difference as

[1] Epidemic Ophthalmia, 1895.

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is here revealed must have some basis in reality. In this country it is well known that the Irish are great sufferers and carry the tendency to the disease with them wherever they go. Among the emigrants from the continent of Europe to these shores the Polish Jews and the Italians (the majority from Southern Italy) form a large contingent of the "trachoma brigade" in the clinics of our large towns. Next after these come the Teutonic races. The native American Indian in the United States suffers severely from the disease. Chibret,[1] however, states on the authority of Dr. Foucher, of Montreal, that the Indians of Canada are practically exempt. The Russian Mennonites living along with them are much afflicted with the disease. The native-born American, but with probably a mixed nationality, in some of the interior middle portions of the United States, as Southeastern Kentucky (Ray) and the mountains of West Virginia (Ayres, Belt), are affected very extensively with a very malignant form of the disease. Among these people there is little, if any, admixture of Italian or Jew, but in many the antecedents were Irish or Scotch-Irish.

There is one race, however, in our heterogeneous population which seems to enjoy an almost complete immunity from trachoma. The race of negroes found in this country seldom or never has the disease. At a meeting of the International Ophthalmological Congress held in New York in 1876, I first called attention to this fact, and the experience of my confrères practising in sections of the United States where the African race abounds has since substantiated this in the main. In an experience of nearly twenty years and embracing many thousand eye cases from this race, I have seen not more than three cases of genuine trachoma, and these were in persons of mixed blood; and even in those cases the diagnosis may not have been certain. I have yet to see a case of entropion in a negro due to trachoma; and, after all, the cicatricial contraction of the conjunctiva is the only certain diagnostic characteristic of the disease.

My attention was first called to this remarkable immunity many years ago in East Tennessee, where a railroad was being constructed on which both negroes and Irish were employed. All the laborers lived in practically the same way; if any difference, it was in favor of the Irish, for it was during the period of slavery; yet the Irish were severely afflicted with what I now know to have been genuine trachoma, while the negroes entirely escaped. There have been some cases of trachoma in the negro reported by practitioners farther south than Washington, and especially on the islands off the Carolina coast. But, even setting aside the likelihood of the confounding of severe follicular conjunctivitis with true trachoma, there is still the possibility of these people having been of a race different from those who have furnished my statistics. Africa is a large country, and we have no reason to suppose that the racial characteristics are more homogeneous there than in Europe, for example. The negroes in Washington and in the surround-

[1] Etude de Géographie ophthalmologique sur le Trachom. Rapport à la Société Française d'Ophthalmologie, 1896.

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ing districts of Virginia and Maryland are mostly the descendants of those the first of whom landed on the James River more than two hundred and fifty years ago. The negroes in other localities may have come from a part of the country very remote from that from which the James River cargoes were brought. The same remark applies, of course, to the reported frequency of trachoma in the negro in Constantinople (Van Millingen), Algeria, and in the countries of Europe where the negro is found. The chief and important point is that trachoma is very unequally distributed as to country and race, and all statistics show it. Different races living under the same social conditions and with the same surroundings are not affected in the same degree. The negroes from whom my clinic is drawn live in as overcrowded and unhygienic quarters as the Irish laborers, yet the one race is a victim of trachoma while the other escapes. The experience of Ray in Louisville, Kentucky, is that the white suffers severely and the negro is free, and so of many others practising in the South.[1] The negro in Cuba also enjoys, according to Fernandez, an almost complete immunity from the disease. Chibret (*loc. cit.*) holds that the Celt of Broca has a comparative immunity from trachoma wherever he may be. He may acquire the disease, but has not the power to communicate it to others. The conclusion, therefore, is inevitable that there is some influence at work aside from contagion and unhygienic surroundings. These latter may, and in some cases undoubtedly do, play an important part in the drama, but they are not the initial acts. The ground must have been prepared before these factors can exert their determining influence. The opinion entertained by Professor Alt and left as a legacy to some of his pupils, that trachoma had its first origin in gonorrhœal ophthalmia, has, it seems to me, no foundation in any fact except that already stated, that a purulent conjunctivitis may develop the disease in one in whom the tendency is latent.

Altitude, according to some (Chibret, Graefe), exercises a decided influence, and it has been asserted that it cannot occur above three hundred metres' elevation from the sea-level. This is true only in a limited degree (Farravelli and Gazzaniga, Reisinger), for it has been found much above that altitude and in otherwise salubrious districts. It is seen at Denver, five thousand feet, and at Colorado Springs, ten thousand feet (Rivers), above the sea-level. It would seem from our present knowledge of the subject that altitude *per se* has but little influence, for some countries, as Ceylon, at the sea-level are particularly free from the disease, and, as above mentioned, high elevations do not always give immunity. Undoubtedly altitude by its beneficial effect on the general nutrition enables the system to resist morbific influences in this as in other forms of dyscrasia, and the other conditions of overcrowding, bad air, etc., are not so usually present there. Where, however, there is at these elevations a great deal of dust or

[1] For further statistics on this point, see paper on "The Racial and Geographic Distribution of Trachoma in the United States of America," by Swan M. Burnett, American Journal of Ophthalmology, September, 1896.

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other agents tending to produce an inflamed or irritated state of the conjunctiva, we have the necessary condition for the development of the disease, and it is found to flourish to a degree. Interior districts which are subject to long droughts appear to be the most prolific ground for the development of trachoma, and often in its most malignant form.

The distinguishing features of true trachoma are the chronicity of its course and the exacerbations and remissions of its inflammatory attacks. These recurring attacks can sometimes be traced to an imprudence of some kind, as an exposure to the irritating influences of dust, smoke, cold, etc., which would be likely to bring on an attack of conjunctivitis. Even the most ardent advocate of contagion would hardly affirm that each of these recurrences of inflammation was due to a fresh infection, and yet often between the attacks the conjunctiva returns to almost, if not quite, its normal appearance, with only a few scars as evidence of what it has passed through.

The disease, as has been stated, is not infrequently confined to one eye of an individual, running its course singly for a number of years without its fellow being implicated, and it is often limited to a single member of a large family whose intercourse is most intimate.

As regards a specific microbe, it has not been positively demonstrated, though many have experimented in that direction. Almost every known germ giving rise to purulent inflammation of a mucous membrane has been found in the discharge of trachomatous eyes. The one that some, as Michel and Sattler principally, consider peculiar to the disease is a small diplococcus, but it has not stood the crucial test of experimentation to the satisfaction of the large body of ophthalmologists. Scherl, of Dorpat (1895), found twenty-four kinds of organisms in the trachoma discharge, and Tschmolossow, in the material expressed from the tissue of follicular conjunctivitis and trachoma, found the staphylococcus pyogenes aureus and albus nine times, the staphylococcus pyogenes citreus four times, and a short Loeffler-like bacillus eight times. Krüdener claims (1895) to have found a kind of plasmodium in the secretions and tissues of the trachomatous eyes not found in follicular conjunctivitis.

FIG. 8.

Trachoma follicle (after Rachlmann), showing the collection of round cells inside the follicle which has a normal epithelial covering. At *a* the cells are undergoing a softening process. Blood-vessels are seen at the base.

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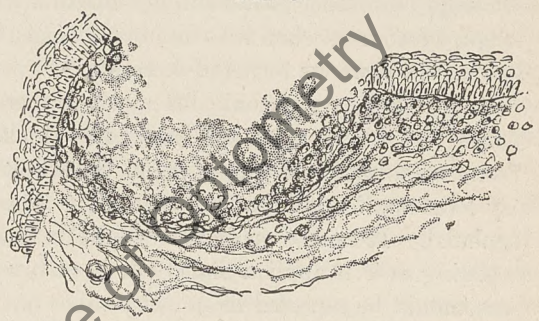
A long war of words has been waged over the nature of the pathologic changes that have been found in the conjunctival tissue the seat of a true trachoma. Is what has been found an entirely new product, as contended by Iwanoff, Berlin, and others, or is it only changes in the normal tissue, as held by Sattler, Jacobson, Reich, Raehlmann, Vincentiis, and others? It seems probable that it is a combination of both.

What seems most likely, in the light of our more recently acquired knowledge, is that the disease has its seat in the adenoid tissue of the conjunctiva, which first undergoes enlargement, with the development, perhaps, of new material in itself and the surrounding tissue, in which an inflammatory process undoubtedly assists. After a certain time this ends in a discharge or absorption of the diseased tissue, leaving a cicatrix as a result. All the adenoid substance may not be, and probably usually is not, affected during one attack; and it seems probable, too, that the disease may be arrested after one or more attacks, as it sometimes is in tuberculosis of the lungs, and no return of the affection experienced. The rule is, however, for these successive returns of inflammation (as indications of a renewed disease of the tissue) to involve in time the whole of the adenoid structure, leading to its final and complete destruction.

An examination of the trachoma granule itself does not show anything histologically distinctive. It contains a gelatinous material and has the composition and character of granulation tissue in general, with small round cells, delicate connective-tissue fibres, and in most instances newly formed blood-vessels (Fig. 8). It is enclosed in a capsule which is rather richly supplied with blood-vessels, and, as has been stated, does not show in its interior any microbe which has been generally accepted as characteristic. Leber in his latest investigations (1896) has found in the contents of the trachoma follicle some large cells containing peculiarly formed bodies which he calls *corpuscle cells* (Körperchenzellen), which are similar to the corpuscles of the lymph follicles of the normal conjunctiva. The condition of the follicle after rupture and at the commencement of the stage of cicatrization is shown in Fig. 9.

FIG. 9.



Trachoma follicle (after Raehlmann). The second stage, showing rupture of the follicle and discharge of its contents.

Treatment.—The therapeutics of trachoma is as varied as are the ideas regarding its pathology. All plans of treatment, however, are directed to causing a disappearance of the trachoma deposit with as little destruction of the normal tissue as possible. At the present time it can be very properly considered under two heads, the medicinal and the surgical.

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The *medicinal treatment* of trachoma consists for the most part in the application to the affected surface of some kind of astringent or caustic substance which will temporarily increase the hyperæmia of the part and in this way hasten the absorption of the morbid material. Caustics are not now used, as they were at one time, to burn off the granulations. This procedure leads to the very state of affairs which we wish to avoid,—namely, a destruction of the normal tissue, with a resulting contracting cicatrix.

It would be safe to say that every known astringent and caustic has been recommended and used at some time as a local application in trachoma, and all with some show of success. It must be remembered, in this connection, that trachoma is essentially a self-limited disease so far as the course of each successive deposit is concerned, and that even under no treatment great amelioration in the symptoms and appearances occur when the deposit has in due course spontaneously disappeared. It must not be inferred from this, however, that trachoma is a field for the display of therapeutic nihilism. There can be no question that much may be done for the relief of symptoms and for the expediting of the natural progress towards resolution. We have, moreover, to deal not solely with the diseased conjunctiva, but also nearly always with an associated keratitis. Fortunately, the treatment of the one is not usually contra-indicated by the other, and our applications to the conjunctiva, even though they be very severe, do not have a deleterious effect on the corneal trouble. On the contrary, as the conjunctiva improves under treatment the keratitis is correspondingly ameliorated, except, of course, when it is due to trichiasis, and this is especially true of the densely pannous forms of keratitis.

In the ordinary chronic form, where there is little if any purulent discharge, and where there is considerable thickening of the conjunctiva, the crystal of sulphate of copper is, on the whole, the safest and most satisfactory local application that can be made. The effect of the remedy is easily regulated. It can be made very slight or severe almost to a caustic action according as it is applied lightly or heavily and repeatedly. The applications should be repeated from every other day to twice or once a week, and the crystal should touch every part of the conjunctiva within reach, not overlooking the retrotarsal folds.

The solid stick of nitrate of silver should never be used under any circumstances, and the mitigated stick (equal parts of nitrate of silver and nitrate of potash) should be limited, if used at all, to cases in which there is a pronounced purulent secretion. The same may be said of nitrate of silver in solution (five to ten grains to one ounce). Bichloride of mercury has been recommended in solution or rubbed as a powder on the surface of the granulations.

There is one remedy which had quite a vogue at one time in the treatment especially of the pannus resulting from or accompanying trachoma, which deserves a passing notice. The powder or watery extract of the

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jequirity bean (*Abrus precatorius*) possesses the power of exciting a violent purulent inflammation with a membranous deposit on the conjunctiva. A form of infusion highly recommended is seventy-five grains of powdered jequirity to three and a half ounces of water. Shake well, and let it stand for an hour, and then apply with a brush. This induced inflammation undoubtedly modifies the course of the trachomatous process, and when it subsides leaves the pannus either much thinner or completely absent. It is very seldom used now, because of its danger to the cornea, which is liable to be affected to ulceration unless the pannus be very dense. The same may be said of inoculation with gonorrhœal matter, employed much in Belgium some years ago. These methods are not without value in very dense pannus, but are two-edged swords.

For the treatment of the keratitis itself there is usually no occasion unless there is an ulceration, when atropine, combined it may be with cocaine, should be used as if it were an independent disease.

Trachoma being something more than a mere local disease and the manifestation of a special dyscrasia, general treatment should by no means be neglected.

The hygienic surroundings of the patient should be carefully looked after, especially in the matter of overcrowding. Since the disease is not contagious, or only very slightly so, isolation of the patient is not usually necessary in the chronic form where there is no purulent secretion. A purulent discharge from the conjunctiva, however, is capable of exciting inflammation of a catarrhal or purulent kind in another healthy conjunctiva, and care should be taken that others do not use the same basins, towels, handkerchiefs, etc. All eyes with a muco-purulent or purulent discharge should be isolated as much as possible. Smoke, dust, and vitiated atmosphere of any kind should be avoided, and the eyes should be protected from bright light by means of blue or gray glasses. Patients should be in the open air as much as possible, on account of its influence on the general health. While a high altitude does not by any means give an immunity from trachoma, there seems to be little doubt that it exercises a most favorable influence on the course of the disease, as it does on all forms of dyscrasia. Where it is possible, the patient should be sent to an elevation of at least three hundred metres above the sea-level, but not to a locality where there is much dust. Out-of-door occupations are not always to be commended, and certainly not those necessitating work amid dirt or smoke or in stables. General farming is not suitable for such patients.

The *surgical treatment* has received much more attention within the last few years at the hands of ophthalmic surgeons. Some form of surgical treatment, in the way of excision of the granulations, has been practised from the very earliest times. This certainly cured the disease, for it removed the morbid tissue, but, unfortunately, it removed most of the normal tissue with it. This method has been revived by Stephenson, who

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excises the upper fornix, he says, with no inconvenient contraction of the conjunctiva.

The more recent surgical methods, and the only ones which should be employed, are *grattage*, *brossage*, and *expression*. A description of the manner of executing these procedures is to be found in the article on Operations, and we shall only briefly point out here some general indications for their employment. Of these methods, that of expression or squeezing is the one to be commended in most cases. It removes the morbid tissue with less injury to the normal constituents than the others, but it is sufficient only in the earlier stages with a limited number of granules and where the conjunctiva is not very much thickened. Where the conjunctiva is thickened and the trachoma granules are hidden under the very much enlarged papillæ, the roller forceps of Knapp or the forceps used by Noyes and others is unable to squeeze out easily the deeply embedded material through the overlying mass. In such cases it is advisable to make horizontal parallel incisions in the conjunctiva before applying the forceps. Brossage and grattage cannot have any advantage in point of efficacy over simple expression or the modification of it just mentioned, and they are certainly more destructive of the normal tissues.

At the present time expression is the treatment *par excellence*, and the local applications only adjuncts and accessories. It is the most rational treatment, too, and the one most in accord with the ideas of the pathology of the disease set forth in this article, and therapeutically the most thorough vindication of them. The morbid tissue is removed and the disease is cured, at least for the time or until a new deposition takes place in another part of the conjunctiva.

Another method of dealing with granulation surgically is to touch each granule separately with the fine point of a galvano- or actual cautery and thus destroy it. This, however, can have no advantage over a properly and carefully executed expression, for it must destroy not only the trachoma granule but also some of the tissue around it, and is at best applicable only to the discrete forms of trachoma, where there is but little enlargement of the papillæ to hide them.

For the dense pannus it has been advised to excise a narrow band, from one to two millimetres in width, from around the base of the cornea (peritomy, Critchett). This cuts off the vascular supply to the new tissue, its vitality is diminished or lost, and the new material is taken up and carried away by the absorbents. The same effect can be obtained in cases of pannus of a restricted portion of the cornea by scraping the surface off with a knife, care being taken not to injure the true corneal tissue. This latter method is more applicable to the pannus of the early stages, which is limited almost altogether to the epithelial layer and is regarded by some as a deposit of the trachomatous material and not as an induced keratitis.

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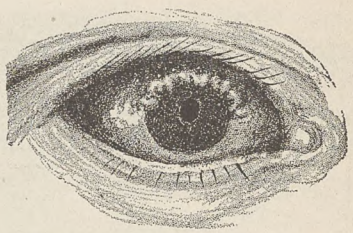
CIRCUMCORNEAL HYPERTROPHY OF THE CONJUNCTIVA (VERNAL OR SPRING CATARRH).

This rather uncommon disease of the conjunctiva was first mentioned by Alt in 1854, and, though its appearance is quite striking, it seems to have escaped attention as a separate affection among the conjunctivitidæ until 1876, when Saemisch again called attention to it in the Handbook of Graefe and Saemisch.

Its characteristic appearance is an elevation around the corneal margin, of an uneven surface, dirty gray in color, varying from one to three millimetres in breadth. (Fig. 10.) It looks not unlike an exaggerated arcus senilis. Its seat of election is the upper margin, though it may show itself at the sides and sometimes forms a broken band around the base of the cornea or completely encircles it. Its seat is just within the sclero-corneal junction, but it nearly always extends, in pronounced cases, some considerable distance on the surface of the cornea itself. There is seldom a pronounced injection of the bulbar conjunctiva, though occasionally it presents an appearance of active hyperæmia in the immediate vicinity of the elevation. An examination of the base of the elevation with a magnifier will nearly always show small capillaries entering into its substance. The cornea inside of this elevation is not affected. The conjunctiva of the lid is always hyperæmic, sometimes slightly swollen, and its surface is covered with very minute granules like dust of meal, though sometimes they are quite large and very hard (Schiele). The conjunctival surface has often the appearance of being covered with a thin layer of milk. In the negro race the bulbar conjunctiva shows a brownish tinge, in some instances very pronounced, corresponding to the palpebral opening, and specks of brown are often noticed in the gray elevation around the corneal base. The conjunctiva of the ball looks thickened and "soggy," and is easily thrown into folds parallel with the corneal base. These latter appearances are also often manifest in the white race.

FIG. 10.



Circumcorneal hypertrophy of the conjunctiva.

The subjective symptoms are those of hyperæmia or congestion. There is a feeling of discomfort, with burning, itching, which is sometimes irksome, and heaviness of the lids, and occasionally there is photophobia. There is seldom any discharge of mucus or pus.

The chief clinical peculiarity of the disease is its appearance with the first warm days of spring or early summer, and its disappearance at the beginning of cool weather, whence the name *vernal or spring catarrh.* In some rare instances, however, the disease continues with diminished intensity during the winter. It makes its appearance again at the beginning of warm weather, and may do so for several years in succession.

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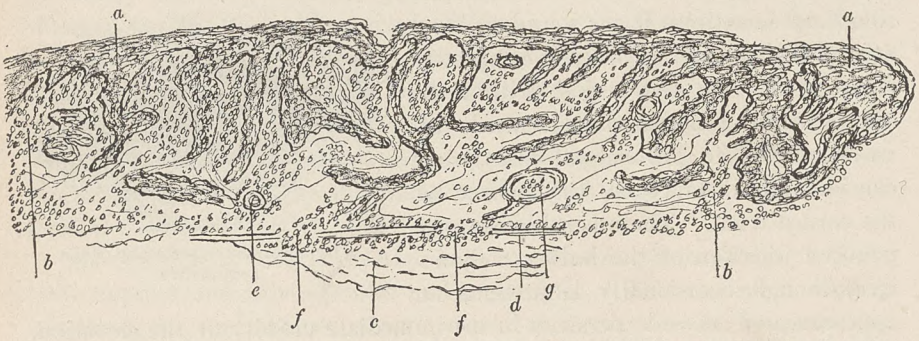
The exact pathology of the disease has not been determined. It appears, however, to be a proliferative and degenerative affection of the epithelial layer of the conjunctiva, including that of the cornea at its base. By some it is considered as of an eczematous nature. The elevation around the base of the cornea consists almost entirely of proliferated epithelial cells arranged in a peculiar manner not unlike the plugs of a cancroid, as shown in Fig. 11. A section of the large granular bodies sometimes found on the palpebral conjunctiva shows a stroma for the most part of lymphoid cells traversed by scanty bands of connective-tissue fibres. The tissue is rather vascular. The epithelium covering it is thickened and runs like pouches

FIG. 11.



Circumcorneal hypertrophy of the conjunctiva. Section through the elevation, showing the peculiar ramification of the proliferated epithelial cells *a* among the tissue composed of round cells and a small amount of connective tissue, *b*; *c*, the cornea; *d*, Bowman's membrane; *e*, a blood-vessel; *f*, break in the membrane of Bowman, allowing the round cells to enter the cornea; *g*, cellular collection in the form of "pearls." (After Schiele.)

into the interior. The anterior layers of the cornea are usually implicated to some degree. No microbe peculiar to it has yet been found.

We sometimes see cases in which the circumcorneal changes are so slight as to escape any but the most careful scrutiny, the appearances of the other portions of the conjunctiva being characteristic and the clinical symptoms typical of the disease. It may be confined to one eye, but usually affects both. When the disease subsides it leaves no trace behind, except a narrow band of slight opacity on the surface of the cornea that can sometimes be detected on close inspection.

Thus far it has not been possible to associate it with any special dyscrasia, though in some cases an enlargement of the glandular system has been noted. It occurs mostly in children and young people, though I have seen it as late as the thirtieth year. It has been thought by some (Chibret) to be an attenuated form of trachoma. Anything like a close connection between the two, however, remains to be proved.

As regards therapeutics very little is to be said. Nothing seems to influence the regular course of the disease. We can only treat the symptoms, which are those of hyperæmia or congestion. Mild astringents and aseptics, as biborate of sodium and boric acid, are best in the way of local applica-

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tions, with frequent bathing of the lids with cold water or some cooling lotion. In case there is a discharge (which is very rare), it should be treated with zinc or nitrate of silver. As the grayish elevation around the base of the cornea is only one manifestation of the disease, its removal by cutting or caustics would avail nothing, though it has been recommended. Aside from a care of the general health and putting the patient in good hygienic condition, general treatment is not indicated, though some have affirmed that the internal administration of arsenic has been beneficial.

Several cases have been reported of a peculiar *reddish-brown succulent thickening of the conjunctiva* surrounding the cornea to an extent of from six to eight millimetres. In most instances the periphery of the cornea is also involved, and in some the process has penetrated the sclera to the interior tissues, followed by loss of the eye. All are not probably of the same origin or pathological character. Rheumatism has been present in some cases, and a suspicion of syphilis in others.

Schlodtmann (1897) has examined one such case histologically and found enormous enlargement of the lymphatics under the epithelium, and the infiltration, in addition to round cells and necrosed tissue, contained many giant cells.

TUBERCULOSIS OF THE CONJUNCTIVA (LUPUS).

The disease described as *lupus conjunctivæ* by the older writers was, without doubt, the same that we now recognize as tuberculosis. The clinical distinction that is made at the present day between the two is that in lupus the disease begins in the skin and passes over to the conjunctiva, whereas in tuberculosis the ulceration is primarily of the conjunctiva. In both the tubercle bacillus is found, and by many it is regarded as essential to the diagnosis.

When it first appears, a portion of the conjunctiva is thickened, with one or more yellowish nodules on the surface, some of which may already be in a state of ulceration. The ulcer has ragged edges and its bottom is uneven and "worm-eaten" in appearance, is covered more or less with pus and broken-down tissue, and may have considerable induration around it. The nodules or ulcerations may occur on any portion of the conjunctiva of the ball or the lids or the retrotarsal fold. They vary in size from that of a pin-head to a destruction of tissue embracing almost the entire surface of the conjunctiva. (See Fig. 1 on Plate I.) When the conjunctiva next the cornea is affected, the cornea itself, even when not involved in the ulceration, becomes more or less opaque. When the disease is extensive and affects the opposing surfaces of the conjunctiva of the ball and the lid, an adhesion between the lid and the ball (symblepharon) takes place during the process of cicatrization. In some instances this has become total. The lids are usually considerably thickened, but are not hard, and there is commonly a rather scanty discharge of a muco-pus. The granular-looking ulcers bleed only on rough handling. There is not much pain complained of except when the cornea is involved. The disease may be limited to one

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eye, but both eyes are often affected, either simultaneously or in succession. The preauricular and submaxillary glands are enlarged on the side corresponding to the affected eye.

There can be no question that tuberculosis of the conjunctiva may be a primary affection and occur in persons in whom there is no evidence of its existence elsewhere. In that case it is due to direct infection, as might easily occur through an abrasion of the epithelium by a foreign body, or through an operation, as that for strabismus, or through the breaking of a phlyctenula. This latter seems the more probable, since the disease is found mostly in children, in whom phlyctenulæ are common. In some rare cases the ulceration passes over the edge of the lid to the integument, but usually it stops short at the inner margin. It might be supposed that the diagnosis of the disease would be easy by a simple bacteriological examination. It is true that in most cases there is no difficulty in demonstrating the existence of the tubercle bacillus either by direct microscopic examination or by inoculation of animals, but there seem to be exceptions. In a case which was under my observation for more than a year an extensive destruction of the conjunctiva in both eyes, one of which was practically lost, presented a typical clinical picture of the disease, and yet I was never able to find a single bacillus after numerous examinations, and inoculation of rabbits gave a negative result. In another instance, however, the inoculation experiment proved the diagnosis after repeated trials had failed. Such cases are usually mild in their course and the infection slight. Clinically I class such cases under tuberculosis, for they present the same appearances and run the same course as those in which the bacillus is found. Bacteriology has not yet become so exact that we can rely upon it solely and absolutely in diagnosis. There can be no doubt that many mild cases of infection pass by unnoticed or are mistaken for more than ordinarily obstinate phlyctenulæ.

The course of the disease is essentially chronic, and the ulcerative process may be arrested short of total destruction of the conjunctiva. It would seem possible that tuberculosis of the conjunctiva might serve as a focus for the dissemination of a general infection. Absolute proof of this is yet wanting, and it is certain that in most cases it remains a purely local affection.

Treatment.—When the nodule or ulcer is small, its extirpation by the knife or destruction by escharotics is indicated as the quickest and safest mode of dealing with it. Of the escharotics, the actual cautery and galvano-cautery are the more radical, while nitrate of silver, pure carbolic acid, and formalin, pure or 1 to 10 or 20, are less severe, can be repeated more frequently, and are more easily modified in their effects.

When the surface affected is of an extent not to permit of such strong measures, the milder antiseptics and aseptic solutions are used locally for cleansing purposes. Among these the best are: formalin, 1 to 1000 or 1 to 2000; bichloride, 1 to 5000; and saturated solution of boric acid, or dusting with iodoform powder.

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As the appearance of the affection may be an indication of the existence of a diathesis or a predisposition to tubercular disease, the general treatment and hygienic management suitable for the condition are indicated and should be carried out.

Leprosy may attack the conjunctiva. Lopez, of Havana (1889), in his studies of this disease as it affects the eyes, finds a conjunctivitis which is the result of traumatism due to the anæsthetic condition of the eye in general which is a part of the disease. There are also frequently pterygia apparently due to the same cause. The leprous tubercles developing on the conjunctiva have the same character as those developing on the skin, and their point of election is the corneo-scleral junction. Bull and Hansen in their monograph on "Leprous Diseases of the Eye" (1873) state that it is rare for the conjunctiva to be attacked independently of the other parts of the eye. They found the cornea the tissue most frequently and characteristically affected, the conjunctiva being only secondarily involved.

SYPHILITIC AND OTHER ULCERS OF THE CONJUNCTIVA.

The specific sore of syphilis may be found on the conjunctiva. It is usually communicated by kissing, and is thus sometimes conveyed from nurses suffering from mucous patches to the eyes of the children under their charge. Infection may also occur by the fingers which have been handling a sore. These sores have the characteristics of specific ulcers on other parts of the body. Where there is a well-marked induration there will be less difficulty as to diagnosis, but there are ulcers which are soft and in which the history of infection is more than doubtful. The preauricular and submaxillary glands are swollen and hard in the true chancre. The doubtful cases have the appearance of the so-called rodent ulcer, but I have found them to heal quite rapidly under the use of large doses of the iodides, even after caustics had been used unavailingly. These as well as the undoubted syphilitic sores occur usually on or near the edge of the lid, though they are sometimes found on the bulbar conjunctiva and the retrotarsal fold. In one case under my observation, a woman of twenty-five years, an ulcer with a rather hard base appeared first on the conjunctiva of the ball near the lower retrotarsal fold, resisted all local applications, and was finally excised. This was followed by perfect healing. A similar ulcer appeared in a few weeks on the conjunctiva of the ball near the outer edge of the cornea in the other eye. This was not excised, but healed in about a month, to reappear on the conjunctiva of the ball near the upper retrotarsal fold in the same eye. She was taking mercury and the iodides under a suspicion of its being specific from the beginning. A histological examination of the excised piece showed nothing beyond an infiltration of round cells.

In the treatment of true syphilitic ulcers no attempt should be made to destroy them with caustics. They will heal rapidly under the internal administration of mercury with local application of antiseptics of moderate strength.

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There would seem to be no reason why *mucous patches* should not occur during the regular course of the disease on the conjunctiva as well as on other mucous surfaces, and several such cases have been reported. One case at least has been reported in which a true *gumma* had its seat on the conjunctiva.

CONJUNCTIVITIS ACCOMPANYING THE EXANTHEMATA.

Like other portions of the air-passages, the mucous membrane of the eye is liable to suffer during attacks of measles, scarlet fever, and small-pox, and sometimes the eye-symptoms constitute a very prominent feature.

The conjunctivitis associated with the exanthemata may be hyperæmic, catarrhal, or purulent, or all three at different periods. The general character of these affections will be found described in the sections devoted to those forms, and requires no additional consideration here. A pustule of *small-pox* may show itself on the conjunctiva, but the most destructive action of this disease is on the cornea. The conjunctiva may also be inoculated with *vaccine matter*.

Very commonly after convalescence from the exanthemata, and in measles particularly, there remains a chronic hyperæmia or congestion of the conjunctiva, which is very persistent and obstinate to all manner of treatment.

Conjunctivitis is very often a most prominent feature in *hay fever*, *rose cold*, and *epidemic influenza*, and frequently demands special attention.

PEMPHIGUS CONJUNCTIVÆ.

In certain rare instances pemphigus has been found to affect the conjunctiva. In some cases the disease has existed simultaneously on other portions of the body, particularly at the angles of the nose and mouth, and sometimes in the throat, but in a few it seems to have been a primary affection of the conjunctiva.

The bullæ, though their form is not usually well defined, are first noticed on the fornix or bulbar conjunctiva. After a time, which may be months, others appear on the ball and palpebral surface, and the final result is an ulceration leading to a cicatricial shrinkage of the entire conjunctival tissue and an adhesion of the lids to the ball through their whole extent (symblepharon), and sometimes of the edges of the lids themselves (ankyloblepharon). The cornea early begins to lose its polished surface, becomes opaque, and participates in the general atrophy. This shrinkage is probably the same as that described under the name *essential atrophy of the conjunctiva*, or *degenerative syndesmitis*. Fig. 2, Plate I., kindly loaned by Dr. Charles A. Oliver, represents well the condition of essential atrophy at a somewhat advanced stage of the disease. Essential atrophy is looked upon by some as an independent affection and not due to pemphigus. The view is gaining ground, however, that all cases of essential shrinking or progressive atrophy begin in an affection of the epithelium of the conjunctiva in the

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nature of an exfoliation. Many of the subjects of this disease are robust. A colored woman of thirty-eight, whom I saw through the kindness of Dr. Belt, of Washington, had never been ill, and the disease came on without warning some three years ago. There is a total symblepharon and commencing ankyloblepharon with a lustreless dry cornea on the right side. The disease in the left eye is not yet so far advanced. There is not that tendency to ulcerative destruction of the cornea which is found in xerosis of the conjunctiva described below.

In many cases it is not easy to demonstrate the previous existence of the perfect bullæ, and in most cases the true bullous character is not apparent, the anterior epithelial layer being ruptured, leaving only a grayish ulcer. The course of the disease is chronic, extending sometimes over a number of years. The treatment is entirely palliative. All operative procedures have utterly failed to give relief.

Some changes in the conjunctiva have been found associated with *herpes iris* in other parts of the body (Arlt).

XEROPHTHALMIA (XEROSIS OF THE CONJUNCTIVA).

Under the term xerosis conjunctivæ are included changes in the conjunctiva which are not all of the same origin. The hardness and dryness of the inside of the lids attendant upon the cicatricial stage of trachoma or other destructive process in that membrane and essential atrophy are sometimes so designated. There is, however, a form of disease which is primary and not associated with any antecedent inflammatory disease. It is characterized by great dryness and dulness of the membrane, in which the cornea sometimes takes part even at the beginning. On the surface of the conjunctiva corresponding to the palpebral opening there is a deposit which looks like fine froth. It is usually in the form of a triangle with its base towards the cornea. This froth is easily removed, but readily forms again. On the edges of the lid there is an accumulation of a thick, white, cheese-like material. The conjunctiva is more or less anæsthetic, and irritation of it does not bring about a flow of tears. In fact, in all pronounced cases the lacrymal secretion is stopped entirely. In severe cases the mucous membrane becomes so shrunken as to obliterate the retrotarsal folds, and assumes almost the appearance of skin. In such cases the corneal surface is very dull, opaque, and often sloughs. A marked subjective symptom which is rarely absent in those old enough to observe it is night-blindness. In a subdued light the patients are practically blind. In nearly all cases of *idiopathic night-blindness* this frothy material above mentioned is found on the conjunctiva. This night-blindness may occur in children—in my cases mostly in young negroes—who seem to be fairly well nourished. It is usually temporary. This milder form has been designated *epithelial xerosis*. It is associated, however, with sclerotic changes in the conjunctiva proper in addition to the degeneration of the epithelium (Basso).

The disease is essentially one of malnutrition, and the most pronounced

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cases are met with in marasmic infants. Cases, however, have been seen in quite robust persons. It has been observed with great frequency on the coffee plantations in Brazil (Gama Lobo), where it seems, as elsewhere, to be sometimes epidemic, and among negro children in South Carolina (Kollock). The frothy material on the conjunctival surface has been found by Basso (1897) to consist largely of degenerated conjunctival epithelium and cells coming from the Meibomian glands, and the emulsion-like substance on the edges of the lids has a similar composition. In this material (which is present also at times in the cul-de-sac) there have been found a number of microbes, and some have thought that there is a specific form which is the producer of the disease; the microbe most commonly found has somewhat the shape of the Klebs-Loeffler bacillus, the so-called *pseudo-diphtheria bacillus*, and is frequently present in large numbers. It seems more reasonable to suppose that these microbes come from the outside, the eyes being partially open most of the time, and find in this degenerated material a very suitable ground for their development, and that they are in no way connected with the production of the disease.

The whole clinical history of the disease shows it to be one of defective nutrition and lowered vitality. That the central nervous system bears the brunt of the burden, the insensitiveness of the retina, as manifested by night-blindness, is strong evidence, though some place the seat of the visual trouble in the cell-layer of the retina. The atrophies of the conjunctiva, the lacrymal gland, and the cornea would appear to be the peripheral manifestations of this central lesion. In infants there is most commonly a fatal issue through intestinal or lung trouble.

Associated with some forms of chronic conjunctivitis and keratitis there is found a localized xerosis of the conjunctiva without night-blindness (*secondary xerosis* (Leber)), which probably is the same as the *plaques épithéliales de la cornée* of Hocquard.

Treatment should be directed to improving the nutrition. Locally asepsis and emollients such as white vaseline are indicated.

LYMPHOMA (ADENOMA) OF THE CONJUNCTIVA.

This is a rare form of conjunctival disease, and may be confounded with certain stages of trachoma occasionally met with or with amyloid degeneration. Indeed, there are many facts which point to the probability of its being the first step in this latter form of conjunctival degeneration. I have met with only two cases which I consider should be properly classed under this head. In certain parts of Europe, especially Russia (Raehlmann), it seems to be frequent.

The conjunctiva, generally of the upper lid alone, is enormously thickened, sometimes to five or six times the normal, and there is an inability to lift it on account of its weight (ptosis). The conjunctival surface is irregular and traced over with deep sulci, but not pronouncedly granular. It is rather hard and gelatinous-looking, and does not bleed easily on rough

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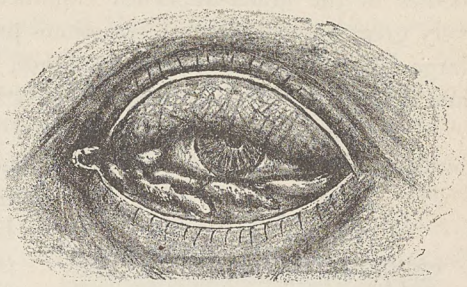
handling. It sometimes affects the lower lid and caruncle and retrotarsal folds, usually in the form of large prominent round swellings like those depicted in Fig. 12. There is little or no secretion, the cornea is usually clear and unaffected, and there is commonly no pain complained of. The disease is almost always bilateral. Michel has found the general glandular system to be enlarged. It occurs most frequently in boys of from seven to eighteen years.

FIG. 12.

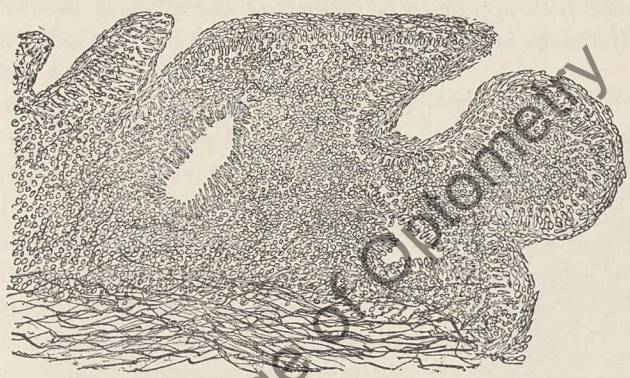


Lymphoma of the conjunctiva.

Wecker has described a form of the disease which affects the conjunctiva around the base of the cornea, looking not unlike an exaggerated form of circumcorneal hypertrophy of the conjunctiva.

Section of the membrane shows it to be composed of enlarged papillæ with hypertrophy of the adenoid structure, but no amyloid degeneration. (Fig. 13.) In one of the cases under my observation there was an exten-

FIG. 13.



Lymphoma of the conjunctiva, showing enormous enlargement of the papillæ covered with epithelium and dense infiltration of adenoid tissue with round cells. (Section of Fig. 12.)

sive colloid degeneration in the deeper portion of the thick mass. The epithelial layer is intact, and in some parts much thickened. The tarsus does not seem to participate in the pathological process, certainly not at the beginning.

It is asserted that there is a tendency to improvement after puberty. The only treatment is operative. In the milder cases deep incisions are commended by Wecker, but where the thickening is great the only thing to do is to remove the mass with the knife and scissors, or with the curette when there is a colloid degeneration. As there is usually a diffuse adenitis, the general health requires attention, and removal to a high dry climate is advisable.

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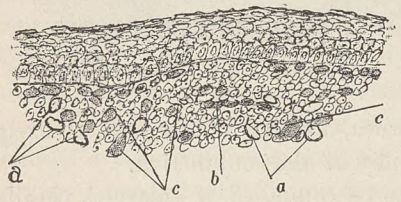
AMYLOID DEGENERATION OF THE CONJUNCTIVA.

This rare form of degeneration of the conjunctiva has been observed principally in Russia. Its chief peculiarity is the presence of amyloid bodies in the much-thickened conjunctival tissue. The hypertrophy is very great, the conjunctiva sometimes protruding from between the lids in large folds. It has a waxy appearance, is smooth and of some consistency, and does not bleed easily when roughly handled.

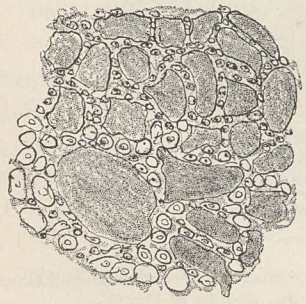
It begins usually in the retrotarsal folds, but successively invades the bulbar and tarsal portions and the caruncle. The upper lid particularly is much thickened, and too heavy to be lifted, and as a consequence there is complete ptosis. The hypertrophied tissue sometimes falls down in folds over the cornea, which latter, however, is not affected. The disease is very chronic in its course, the period of its growth extending generally over many years. There is no discharge or lacrymation, and no pain. It has been thought by some to be a sequela or a very exaggerated or modified form of trachoma, which seems possible, since both appear to be diseases of the adenoid tissue of the conjunctiva; but there is no doubt that it can be an entirely independent affection and strictly local in its manifestation without the concomitant appearance of the same kind of degeneration in any other part of the body. It is Raehlmann's opinion that the amyloid bodies are seen only in the later stages of the peculiar degeneration, and that their presence is not necessary for a diagnosis of the disease. The first step is a simple hypertrophy of the conjunctiva, an increase in its adenoid elements, such, for instance, as that described as lymphoma in the preceding section. The next step is a hyaline degeneration, and the final one is the appearance of the amyloid bodies. (Figs. 14 and 15.)

Fig. 14.



Amyloid degeneration of the conjunctiva, early stage: *a*, hyaline degenerated cells; *b*, first appearance of amyloid bodies; *c*, very pronounced amyloid degeneration. (After Raehlmann.)

Fig. 15.



Amyloid degeneration of the conjunctiva, advanced stage. The larger bodies are the amyloid masses, the smaller are epithelial and round cells. (After Raehlmann.)

The only treatment is operative, and Raehlmann has found that a partial extirpation is almost always followed by atrophy and final disappearance of the remainder of the hypertrophied mass.

CHEMOSIS OF THE CONJUNCTIVA.

The loose texture of its tissue easily makes the conjunctiva the seat of effusions of various kinds. One of the most common of these is that of blood-serum, causing the condition known as œdema, or *chemosis*, in which

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the cornea is seen as if in the bottom of a crater-like elevation of the conjunctiva surrounding it. It may accompany inflammatory conditions of the conjunctiva itself, or of the iris or the ciliary body, and is sometimes a prominent symptom in these conditions. It is found in acute glaucoma, in affections of the orbit, and sometimes in those of the lids. It is always indicative of an obstruction to the return flow of the circulation. The degree of inflammation is not always indicated by the amount of chemosis, for it frequently happens that there is a large amount of effusion with but slight pathological change. The condition of the vascular walls has undoubtedly much to do with the origin and the amount of effusion. Sometimes there is a chemosis without any inflammatory symptoms whatever. This occurs usually in old persons, and is probably due to some temporary local interference with the circulation, the so-called *idiopathic chemosis.*

We occasionally find a chemosis coming on suddenly in apparently strong and healthy persons, due to some disturbance at the nerve-centres the result of a toxic agent. I have seen a marked chemosis of the conjunctiva and lid following the ingestion of a single grain of quinine. Every time quinine in any appreciable quantity was taken this result followed. In early life an attack of urticaria followed the administration of quinine, showing that the drug had in this instance a peculiar influence on the vasomotor system at the periphery. Burning and itching of the conjunctiva, with hyperæmia, have been noticed, with the same condition of the face, in attacks which were probably *urticaria* and caused by the ingestion of food which had before produced general urticaria.

A pure chemosis is characterized by a uniform transparent thickening of the conjunctiva, principally of the ball. It is of a pale-pink or yellow color, in contradistinction to the deep red of the blood effusion of ecchymosis.

The subconjunctival tissue stopping short at the base of the cornea, the spread of the effusion is arrested there and rises up around it like a mound, with the cornea at the bottom, whence its name (χήμη). The swelling following the bite of an insect or a bee-sting is a typical form of this affection.

There is also an œdema called "filtration chemosis," which comes from the oozing of the aqueous humor from the anterior chamber through a small opening at the sclero-corneal margin. The chemosis which attends upon inflammation is sometimes called "inflammatory œdema," to distinguish it from the forms not dependent upon hyperæmia or congestion.

Since it is usually symptomatic, there is, as a rule, no call for treatment addressed directly to the condition itself. Occasionally, however, the swelling is so excessive as to endanger the nutrition of the cornea by its pressure, and it is then necessary to evacuate the liquid by puncturing the conjunctiva. This may be done under cocaine by a needle or by making a number of small cuts with the scissors.

To facilitate the absorption of the fluid, compresses of lead and laudanum or hot boric acid may be used.

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ECCHYMOSIS OF THE CONJUNCTIVA.

Rupture of a blood-vessel of the conjunctiva is followed by an extravasation of blood in and under its tissue, giving rise often to a very startling appearance. However circumscribed the effusion may be at the outset, it gradually spreads on account of the looseness of the conjunctival and subconjunctival tissue, and sometimes, when the amount of exudation is large, will cover the whole anterior surface of the ball except the cornea.

Its color at first is deep red, but as absorption goes on it becomes more yellowish, and finally disappears altogether, rarely leaving any mark. The breaking of the vessel may depend upon a direct traumatism of some kind, as a blow, or may be caused even by violent rubbing of the eyes. It follows often upon some effort during which the blood is forced to the head, as straining at stool, hard vomiting, coughing, etc. It is very common in children suffering with whooping-cough, and is frequently seen among the aged and those whose vascular walls are weakened from any cause, and may indicate a general atheromatous condition of the blood-vessels.

The ecchymoses which appear in the conjunctiva after injuries to the head not involving the eyes indicate the possibility of a fracture at the base of the skull.

When the extravasation is fresh it is sometimes possible to express some of the blood through small incisions in the conjunctiva. Usually, however, it must be left to the absorptive powers of nature. This may be assisted by an application of the lead and opium solution, hot boric acid solution, and mild massage through the closed lids.

EMPHYSEMA OF THE CONJUNCTIVA.

The appearance of air under the conjunctiva indicates a communication between the subconjunctival tissue and some one of the pneumatic cavities around the orbit, and is usually the result of traumatism. This traumatism may have ruptured the bony walls which divide the contents of the orbit from the nasal cavity, the frontal sinus, or the ethmoidal cells, thus allowing the air from them to enter and diffuse itself through the loose tissue of the orbit, the lids, and the subconjunctival space. It may also follow ulcerative destruction of these bones, leading to an opening in their walls. It is nearly always noticed immediately after a blowing of the nose. The feeling of crepitation under the finger makes a mistake in diagnosis impossible.

A compressive bandage is the only treatment required for the relief of the condition itself. The patient should be cautioned against blowing the nose violently.

PINGUICULA.

A small, usually round, yellowish elevation is often seen on the nasal side of the conjunctiva over the insertion of the rectus internus near the cornea. It is sometimes seen to the outer side of the cornea, and occasionally on both sides. It is most common in mature persons and in those exposed to influences irritating to the conjunctiva. While it does not, as a

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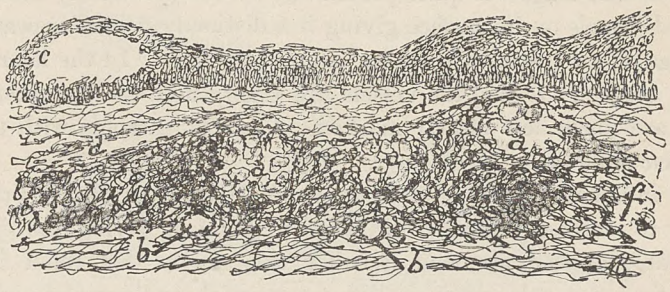
rule, give rise to any trouble, it is thought by some to be the starting-point of a pterygium. This, however, is very questionable. Though it has every appearance of a fatty tumor, there is no fatty matter in its substance. It consists of condensed fibrous tissue and thickened epithelium, and, according to Fuchs, of a hyaline degeneration of the conjunctiva and subconjunctival tissue in which the epithelium takes only a secondary part (Fig. 16). It is thought that an arthritic dyscrasia predisposes to the development of such growths. The active cause is considered to be a local irritation from dust, smoke, etc., but pinguiculæ are often observed in persons who are not exposed to these influences.

FIG. 16.



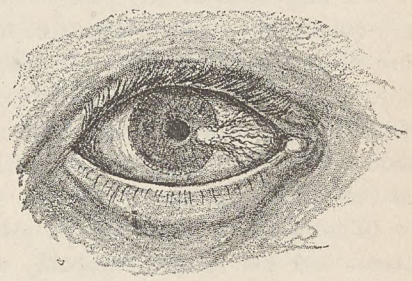
Histological structure of pinguicula. The epithelial layer of the conjunctiva is irregular and much thickened, and there is a separation in the layers at *c*. Hyaline degeneration is shown in the well-defined areas marked *a*; *b*, cross-sections of blood-vessels; *d*, longitudinal section of vessels; *e*, subepithelial connective tissue. (From specimens prepared at Lionel Laboratory of Emergency Hospital, Washington.)

There is usually no occasion for interference, but when there is, the tumor should be removed by the knife or scissors. Local applications are of no avail.

PTERYGIUM.

A pterygium is a flat, pointed elevation on the eyeball, its apex resting on the cornea, its base spreading out over the conjunctiva towards the equator of the ball. Anatomically it is divided into three parts: the *head*, a round, yellowish-white elevation lying on the cornea; the *neck*, the short constricted portion back of the head; and the *body*, which forms the expansion of the growth. It is usually situated to the nasal side of the cornea, but is sometimes found to the temporal side, and on rare occasions on both at the same time (double pterygium). Cases of upward, downward, and oblique pterygium have been reported, but they are uncommon, and but few of them probably are true pterygia. The head may lie on the

FIG. 17.



Pterygium.

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corneal surface anywhere from the sclero-corneal margin to the centre. It rarely passes beyond this. It always starts at the corneal margin, and when it passes beyond does it by gradual encroachment. Its growth is usually very slow, taking years sometimes to reach its maximum size, after which it either remains stationary or shrinks somewhat, becoming less vascular and more condensed in structure. This has caused pterygia to be divided into *vascular* (*P. crassum*) and *membranous* (*P. tenuis*), though these are really only different stages in the development of the same growth. In the vascular stage it is quite prominent, with rather clearly defined edges, the blood-vessels on its surface giving it a distinctly veined appearance like the wings of an insect, whence its name (πτερύγιον). In the later or membranous stage (*P. tenuis*) the vessels are scanty and the limitations of the growth are much less clearly marked. Cases of pterygia with two heads have been reported.

Any inflammatory affection of the conjunctiva, from cold or other cause, is likely to increase this vascularization very much, giving the eye a very disreputable appearance; and it is for this reason that relief is usually sought, for, as a rule, pterygia are not painful, and interfere with vision seriously only when the head covers the pupil.

The etiology of pterygium is obscure. Its prime cause, however, is most likely some form of mechanical irritation. It is found almost exclusively in those who live exposed lives out of doors or are subjected to the irritation of dust, smoke, etc. It is seldom seen in women, and not often in young people. It is thought by some to begin as a pinguicula. By others it is held that an ulceration at the sclero-corneal junction is the commencement of the process. A fold of the conjunctiva is caught in the ulcer; the cicatrix makes traction on the loose membrane and throws it into folds. The process is continued and increased as the cicatrix (the head) advances towards the corneal centre. What causes the progression of the cicatrix horizontally across the cornea is not explained. This progression over the cornea is not seen in those forms of false pterygia which result from burns or ulceration caused by diphtheritic conjunctivitis, etc., and which may occur at any part of the globe. The upward, downward, and oblique forms of pterygia are due most probably to causes of this kind, and are forms of *false pterygia*. In false pterygia there is not that clear division into head, neck, and body which exists in the true pterygium. In the true form the point of a probe passed under the neck comes out on the other side with a fold of conjunctiva pushed in front of it, showing the adhesion at that place to be very loose.

The fact that true pterygia are nearly always seated over the internal rectus has led some (Theobald) to suppose a connection between their development and the effort at convergence; yet pterygia are not met with most frequently in those who habitually use their convergence.

A microbic origin has been assumed by others, but most probably the association of the two is a mere coincidence.

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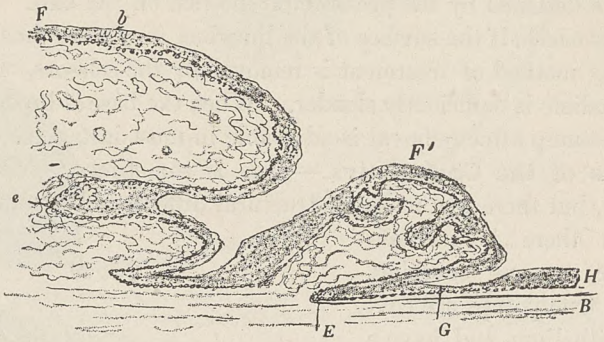
An examination of the conjunctival part of the growth shows an increased quantity of fibrous tissue, considerable cellular infiltration, and an increased number of blood-vessels. The head of the pterygium is embedded in the tissue of the cornea, and often the membrane of Bowman is destroyed, and always it is affected in such manner that the corneal tissue never regains its transparency after removal of the growth. The epithelium of the cornea passes down under the head for a distance, and then, turning

FIG. 18.



Pterygium (after Fuchs). *F*, principal fold, *F'*, secondary fold of the conjunctiva; *e*, epithelium filling the space between the folds; *G*, conjunctival epithelium passing over to *H*, corneal epithelium; *B*, Bowman's membrane, reaching to *E*.

upon itself, passes up over the head and covers it (Fig. 18). There are always a few fine capillaries in the head and the corneal tissue adjoining it.

Removal is the only treatment. This may be by ligature, excision, or transplantation. If the tissue is not thoroughly removed, there is a liability to a recurrence of the growth in some degree. (For the details of these operations, see article on Operations.)

TUMORS AND MORBID GROWTHS OF THE CONJUNCTIVA.

The morbid growths which occur in the conjunctiva are those found in mucous membranes in other parts of the body, though some of them have a greater seriousness and significance on account of their situation and proximity to important tissues than they would had they occurred elsewhere. It has been customary to divide them, clinically, into benign and malignant.

Of the *benign* growths the so-called *granulation tumor* is the most common. It accompanies the healing of various wounds and injuries of the conjunctiva, and is very common after the operation for strabismus, particularly when section of the tendon has not been made subconjunctivally, though even in this case the button of granulation springs, partly at least, from the stump of the divided tendon. It is also seen as a button at the bottom of the orbit after enucleation. Foreign bodies embedded in the conjunctival substance, particularly in the cul-de-sac, cause these granulations to spring up around them, and the bleeding from their surfaces gives rise to the phenomenon of *bloody tears*, of which occasional mysterious cases are reported, and at least one case has been reported in which death resulted

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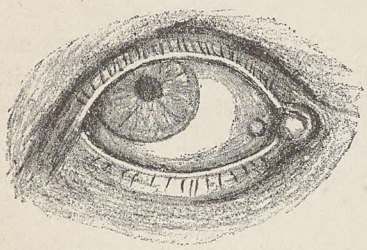
from the long-continued hemorrhage. It is found on the inside of the eyelids at the seat of a chalazion when it has broken through its inner wall and discharged into the conjunctival sac.

Granulomata have the appearance of granulations in the mucous tissues elsewhere; they are soft at the beginning at least, irregular on the surface, and have broad bases. They gradually become pedunculated and smoother on the surface, and sometimes, when the pedicle gets very slender, are rubbed off by the action of the eyelids. When situated on the inner surface of the lids they are flattened by the pressure of the lids on the ball. I have seen them occupy one-half the surface of the lid when they were thus spread out.

The only method of treatment is removal by the scissors, or by torsion when the pedicle is sufficiently slender. When the base is broad, cauterization of the stump after removal is advisable in most instances.

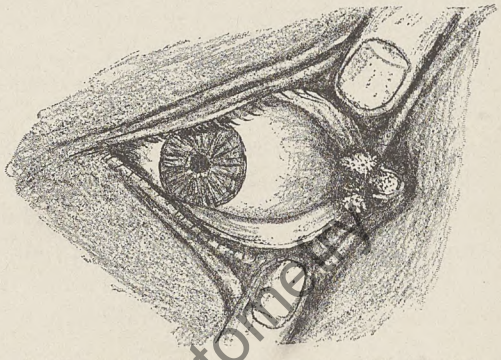
Polypus of the Conjunctiva.—This is sometimes confounded with granulation, but there is a marked structural difference between them. In granulation there is no epithelium or mucous covering, whereas all true polyps are covered by epithelium and have a

FIG. 19.



Polypus of the conjunctiva.

FIG. 20.



Papilloma of conjunctiva at caruncle. (After Steulp.)

smooth even surface. Their substance is composed of connective tissue and blood-vessels. This form of tumor is rather uncommon on the conjunctiva,—in fact, is denied by some observers,—and when found is usually situated over the caruncle. They are small, seldom exceeding the size of a pea. (Fig. 19.) They are very vascular, and bleed easily. They require the same treatment as granulations.

There is a form of polypoid growth called *papilloma*, or sometimes *soft fibromata*, which differs from the one just described in being larger and usually with an uneven surface like a wart and having a structure similar to that of a condyloma. It grows by preference from the caruncle (Fig. 20), but is found in the palpebral conjunctiva also. In typical cases the structure is truly papillomatous,—a central vessel for each papilla, with some connective-tissue elements, but mostly composed of epithelium, arranged quite regularly in layers, squamous on the surface and columnar in the deeper parts.

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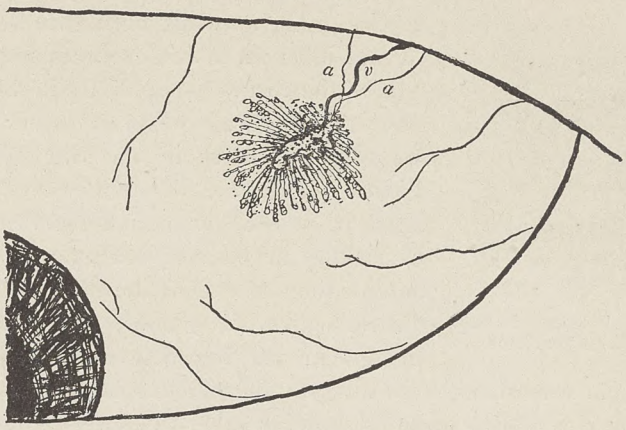
One case differing somewhat from this typical form I have seen on the conjunctiva of the ball of a negro girl of eight years (Fig. 21). It was

FIG. 21.



Unusual form of papilloma of conjunctiva, regular and symmetrical arrangements of vessels ending in loops around the central stem: *a*, *a*, arteries; *v*, vein, entering the pedicle of the growth.

discovered suddenly some months before she came under observation. The formation of new blood-vessels in its substance made a strikingly beautiful picture under a magnifier. It did not bleed on handling. Its structure as revealed under the microscope after removal by cutting its thin pedicle is shown in Fig. 22. Its connective-tissue elements are very scant. The

FIG. 22.

Papilloma of conjunctiva—histological structure section of above: *d*, large vessel entering the base of the growth; *v*, section of blood-vessels. The epithelium which constitutes the bulk of the tumor is shown to be squamous on the surface and columnar near the base and in the deeper parts.

cellular element is entirely of the epithelial variety, and there is a pronounced tendency to stratification, it being columnar at the connective tissue which forms the pedicle and passing gradually to the squamous form on the surface. There had been no injury nor any kind of inflammation to which the origin of the growth could be attributed. Hirschberg and Magnus, among others, have each reported cases somewhat similar. There may be a number of the growths attached at different places to the conjunctiva of the ball or of the lid. A suspicion of syphilis existed in some cases.

It demands the same kind of treatment as the polyp.

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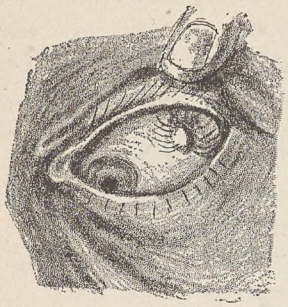
Dermoid Tumors.—Dermoid tumors on the conjunctiva are congenital and nearly always associated with some other malformation or defective development of the eye, particularly coloboma of the lids, or of other parts of the face, as harelip. Their seat by preference is the sclero-corneal junction, and in such cases the conjunctiva of the cornea is involved in the growth. They may occur, however, at other places on the conjunctiva, as in the case reported by Ficino. (Fig. 23.) They were first described by Ryba in 1853, who gave them the name by which they have since been known. They contain all the elements of the skin, including hair follicles, sebaceous glands, and sweat glands, though in varying degrees and proportion. They vary in consistence according to the amount of fatty matter they contain, sometimes being quite hard and at other times very soft.

FIG. 23.

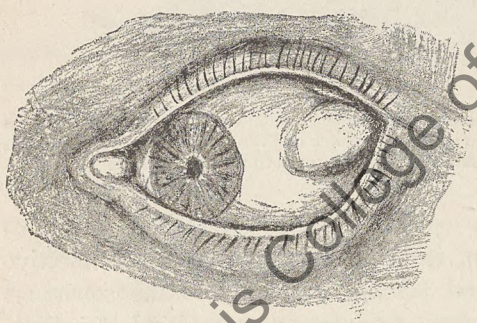


Dermoid of conjunctiva with cilia on its surface. (After Ficino.)

On the surface, which is like that of the integument, there is nearly always hair, usually fine, but occasionally quite large and long. Sometimes they increase in size after birth. The lower external edge of the cornea is their seat by preference, and they vary in size from that of a small pea to more than double that magnitude. Some cases have been reported in which the tumor covered the entire cornea. They demand removal principally for cosmetic reasons and on account of the inconvenience they cause. The excision should be thorough, as there is a tendency to reproduction if the whole tissue be not removed.

Van Duyse has reported one case of *dermo-epithelial tumor* of the conjunctiva situated some distance from the corneal border in a child of four and a half years. It was confined to the epithelial layer, the substantia propria being intact. It was composed of epithelial cells arranged in alveoli. It was not considered malignant.

FIG. 24.



Lipoma of the conjunctiva.

Lipoma of the Conjunctiva.—A growth composed of fatty matter with a small quantity of connective tissue is found under the conjunctiva, usually towards the outer canthus, between the superior and inferior recti muscles. (Fig. 24.) In very rare instances there may be more than one tumor. Though always congenital, lipomata of the conjunctiva frequently show a tendency to increase in size about the age of puberty. They are usually somewhat triangularly pyramidal in shape,

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the base being backward. The surface next the ball is sometimes slightly concave. In size they may reach two centimetres in length by one centimetre at the base; as a rule, however, they are about half that magnitude. They are movable on the globe, and the conjunctiva is generally movable over them and usually is unchanged in character. In some cases, however, the conjunctiva is thickened and seems to have participated in the morbid process.

Lipomata have been described which, besides fatty matter, contained some elements of the skin. These may be called *dermo-lipomata.*

Removal of these growths, which is called for when they become so large as to be inconvenient or unsightly, is easily accomplished through an incision in the conjunctiva, from which and the surrounding tissue they are to be carefully dissected.

Abscess of the Conjunctiva.—Occasionally a circumscribed collection of pus takes place in the conjunctival tissue independent of trauma or other morbid process. It is more apt to be found in the caruncle, which, being somewhat dermoid in structure, is the more likely to be the seat of an idiopathic abscess. I have seen one case, however, in the conjunctiva of the ball to the outer side between the external and superior recti. There was no history of injury, nor were the surrounding tissues of the orbit or eyeball affected. Hot applications and an early opening are the indicated therapeutics.

Cysts of the Conjunctiva.—Simple serous cysts of the conjunctiva are not common, and are not all of the same origin. Some follow traumatisms so closely that the two must be regarded in the light of cause and effect. Others are, no doubt, congenital, and when situated on the sclero-corneal junction partake so much of the nature of dermoids that they might be placed in that category. These cysts contain a clear liquid, sometimes having suspended in it nucleated cells, resembling epithelium.

Rombolotti (1895) describes one in which three cilia were found in the walls of the tumor. Uhthoff (1878) also found cilia in the walls. All these followed injuries, and were very analogous in their etiology to the cysts on the iris which follow the entrance of cilia into the anterior chamber.

Another variety is due to the enlargement of the acino-tubular glands of Krause. These are situated near the retrotarsal fold and on the caruncle, where these glands are most abundant. These and those due to trauma are beneath rather than in the conjunctival substance, and are not movable on the ball to the same extent as those in the conjunctiva itself. Another variety has its origin in enlargements of the lymph-channels of the conjunctiva, an exaggerated form of lymphectasia. It is possible that some of the cases following traumatic injuries are of this latter variety, the injury and the subsequent inflammation blocking up the lymph-channels. Under this head will also fall those cases which have been reported as due to stings

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of insects. In these cases, as well as in those of enlarged tubular acinous glands, there is a distinct lining of the cyst-wall with cells. (Fig. 25.)

Fig. 25.

Cyst of conjunctiva from enlargement of lymph-channel: *a*, epithelium; *b*, endothelium of lymph-space, anterior wall; *c*, endothelium of posterior wall; *d*, *e*, *g*, cellular contents of the cyst; *f*, section of blood-vessels. (After Antonelli.)

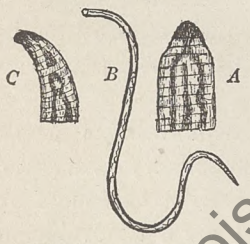
It is to be remembered that there are *false cysts*, simple circumscribed elevations of the conjunctiva by the aqueous humor that has oozed from a fistula at the sclero-corneal junction.

The sole treatment of cysts of the conjunctiva is ablation. Usually it is necessary only to take away the anterior wall. Recurrences are uncommon.

Osteoma of the Conjunctiva.—Very rarely tumors, osseous or fibro-osseous in their structure, are found in the conjunctiva (Critchett, Snell). Their usual seat is on the ball and near the external commissure. As they are observed mostly in young people, it is a fair inference that they are congenital, of the same nature as dermoids, and due to faulty or abnormal development.

Cysticercus under the Conjunctiva.—The cysticercus cellulosæ occasionally finds its home under the conjunctiva. If seen at the beginning it shows itself as a transparent cyst in the interior of which the head of the animal may sometimes be seen. There is more or less vascularization of the overlying conjunctiva, which becomes thickened and opaque in time, and then the diagnosis is difficult. The usual seat of the cyst is on the ball and to the nasal or temporal side of the cornea. More rarely it is found on the retrotarsal fold or the palpebral conjunctiva. It is easily removed through an incision in the conjunctiva.

Fig. 26.



Filaria medinensis. *B*, the whole animal, magnified two times; *A*, its head; *C*, its tail, magnified six times.

Another form of entozoon which is occasionally met with in the conjunctiva in hot countries is the **filaria medinensis.** (Fig. 26.)

It sets up a severe irritation, ending usually in the formation of an abscess. It is from twenty-five to thirty millimetres in length, and enters

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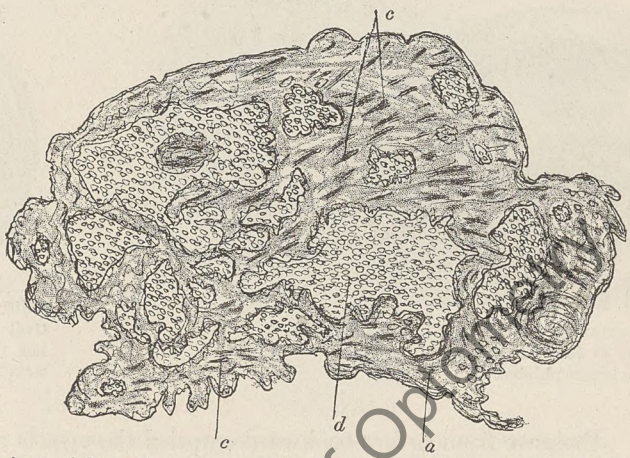
the conjunctiva from the orbit. Evacuation of the abscess carries the worm out with its contents.

Angioma and Telangiectasis of the Conjunctiva.—A nævus of the lid is sometimes carried over and affects the conjunctiva. More rarely there is a true angioma of the conjunctiva alone. The caruncle is the usual seat of these growths, and they appear as soft red tumors with rather sharply defined limits. They are congenital, with a tendency to increase in size, sometimes quite rapidly.

Tumors in which the vascular tissue was predominant have been reported (Rampoldi, Kraschinsky, Stefanini) as springing from other portions of the conjunctiva. It is a question whether such growths are not really papillomata or soft fibromata with an unusual development of blood-vessels. In one case, however, reported by Bossalino and Hallauer (1895), the tumor was found on examination to be a pure muscular angioma of the subconjunctival tissue. (Fig. 27.)

FIG. 27.



Angioma of the conjunctiva: *a*, wall of the tumor; *d*, spaces filled with blood; *c*, muscular fibres. (After Bossalino.)

Prompt treatment is indicated. In one case of nævus of the caruncle which fell under my observation I tried the galvano-cautery, with the effect of diminishing the size of the tumor. The patient disappeared before treatment was ended. This I prefer to excision, as it involves less destruction of the normal tissue.

I have seen one case of genuine *hæmatoma* of the conjunctiva. The eye was much diseased in its interior as the result of an injury, though there was no rupture of the coats of the eyeball that could be discovered at the time. In the course of some weeks a dark-red swelling began to show itself in the conjunctiva to the outer and lower portion of the ball and half a centimetre from the corneal edge. This increased slowly in size, and at the end of a month or so had attained nearly the size of a pigeon's egg. The inflammatory symptoms were not severe, but, as the eye was painful and

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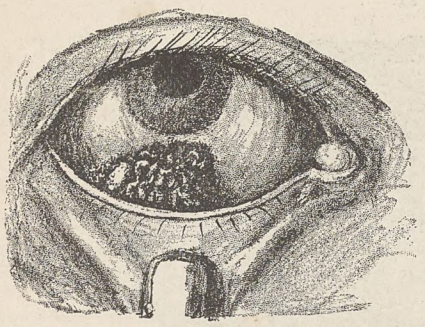
there was a probability of some malignancy in the growth, the eye was enucleated. On examination the tumor was found to be a cyst filled with blood and communicating with the interior of the eye by a small opening in the sclera. The blood came from the vitreous chamber, which was filled with it. The walls of the cyst (which seemed to be in the substance of the conjunctiva) were very thick, apparently from inflammatory exudation.

Tortuous and enlarged veins on the conjunctiva, with no other inflammatory symptoms, are not uncommon as a result of previous disease, such as glaucoma, or of a general vascular change, as in alcoholics.

Occasionally, however, we meet with *true varix* of the conjunctival veins. The three cases that I have myself seen were all found on the ball near the lower retrotarsal fold, and were not associated with any present or past inflammatory affection of the eye. They appeared as a pyramidal bunch of blue veins with the apex towards the cornea (Fig. 28, Plate II.). Pressure from before backward emptied the vessels completely. With this indication I ligated the bunch at the base, with the effect of causing the tumor to disappear permanently. Conjunctival varix has been observed on other parts of the ball. In one of the cases seen by me (Fig. 29) the mass of veins contained two *phleboliths*, one two millimetres in diameter, the other six millimetres in diameter. The surface of these bodies was perfectly smooth, and they were as hard as shot. They were decalcified, and on section their laminated structure was beautifully shown. (Fig. 30.)

FIG. 29.



Varix of conjunctiva containing phleboliths. The light spot to the temporal side marks the location of the larger phlebolith.

FIG. 30.

Section of phlebolith of conjunctival varix, showing the laminated structure.

Lymphectasia of the Conjunctiva.—The flow of lymph in the lymph-channels of the conjunctiva sometimes becomes obstructed, causing the walls of the lymphatics to stand out like small transparent beads on the conjunctival surface. (Fig. 32.) This is nearly always in the palpebral fissure and about midway between the cornea and one of the canthi, though Hirschberg has reported a case in which the beads made circles around the cornea. Leber reports a case of a woman in which the lymph was mixed periodically with the coloring matter of the blood,—*lymphectasia menorrhagica.*

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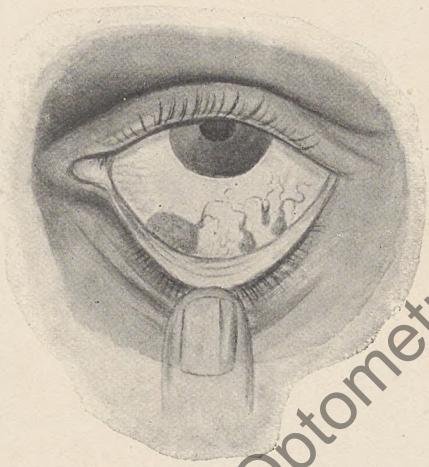
PLATE II.

FIG. 28.



Varices of the conjunctiva. (From a drawing by Dr. McDonald.)

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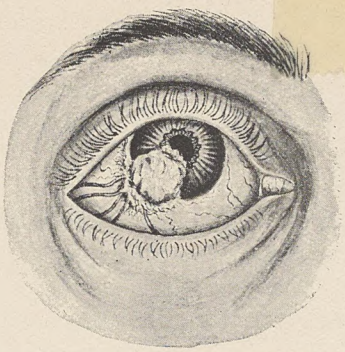
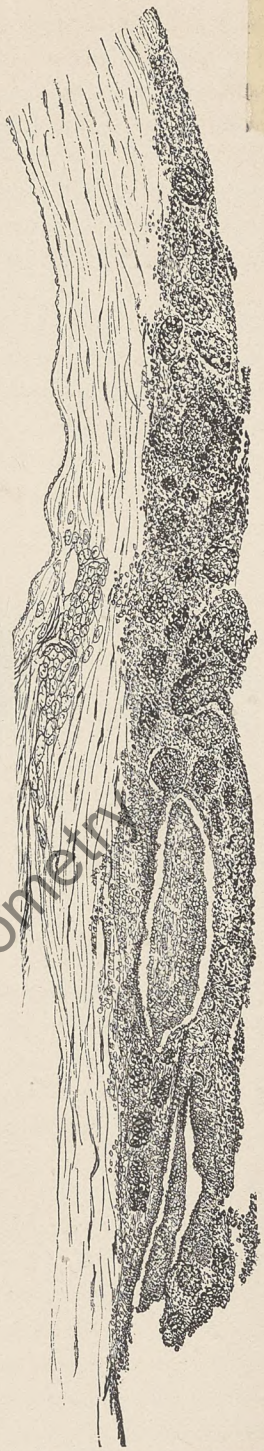
PLATE III.

1.



Macroscopic appearance of the tumor at the corneo-scleral junction. (Oliver.)

2.



Microscopic section showing position and extent of the growth. (Oliver.)

3.

Microscopic section showing infiltration of epithelial cells into the corneal lamellæ. (Oliver.)

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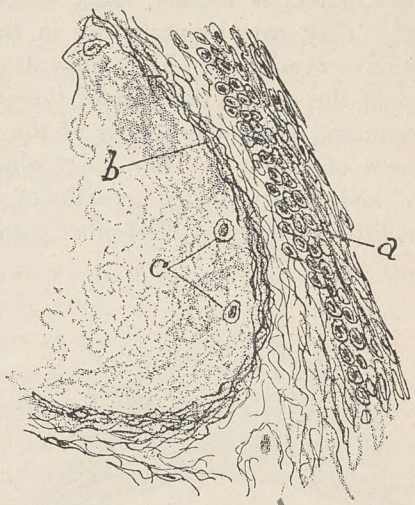
As a rule, they give rise to no trouble. They are sometimes congenital. An enlargement of the capillaries is commonly noticed among the mass; otherwise there is no change. They can be evacuated by pricking each globule with a needle, or the whole may be extirpated if the mass is not large.

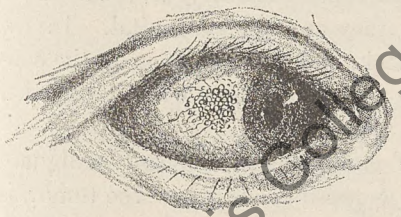
Malignant Tumors of the Conjunctiva.—Like other mucous membranes, the conjunctiva may be affected with malignant growths. Among one hundred and thirty-seven malignant epibulbar growths, Noyes (1879) found seventeen to spring from the conjunctiva and thirty-one from the limbus. By far the larger number of these are of the form known as epithelioma. In fact, some, as Panas, maintain that the so-called sarcomatous forms are really epitheliomata of more rapid growth. Strouse, however (1897), tabulates cases of true sarcoma of the limbus. Most, if not all, of these started in the conjunctiva, and where the cornea was invaded it was only in the epithelial layers or the layers of the substantia propria contiguous. (Figs. 33 and 34.) The case figured on Plate III. is from Dr. Charles A. Oliver's practice, and is a good example of the epitheliomatous form at the sclero-corneal margin. In this case, after several extirpations, the eye had to be sacrificed.[1] The

Fig. 31.



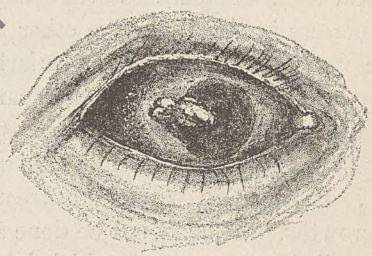
Lymphectasia of the conjunctiva: section of an enlarged lymph-duct, showing, *a*, conjunctival epithelium; *b*, lining of the duct; *c*, lymphoid cells in the contents of the duct. (Panas.)

Fig. 32.



Lymphectasia of the conjunctiva.

Fig 33.



Melanotic epithelioma of the limbus. (Panas.)

variety of melanotic tumors which have been described owe their pigment, Panas thinks, to their situation near the uveal tract or to hemorrhage, and should not be considered on that account as sarcomatous in

[1] Archives of Ophthalmology, April, 1897.

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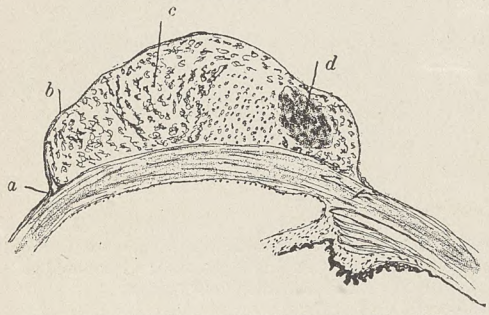
the true sense. Some cases of what seemed to be true carcinoma have been described as occurring on the conjunctiva. Except when they pass over from the integument of the lid to the conjunctiva, these tumors are most commonly found at the sclero-corneal margin. As pointed out by Fuchs, this situation is the analogue of the predilection of epitheliomata for the boundary between two kinds of epithelium, as the edge of the lips, the anus, etc. The locality of the sclero-corneal margin is very subject, too, to proliferation of epithelium of a benign character. The growth of these tumors is usually slow, but they may attain in time to a large size. They grow by preference in the direction of the cornea, sometimes entirely covering it. They may also extend by ulceration into the interior of the eye at the edge of the cornea. The sclera, on account of its toughness, resists the attacks of the growth much longer than the other tissues of the eye. They affect almost exclusively persons advanced in life, and, like epithelial growths of the face, remain localized and show but little tendency to affect other parts of the body by metastasis.

FIG. 34.



Melanotic epithelioma of the limbus (section of Fig. 33). The growth starting from the conjunctiva of the limbus is confined to the epithelium of the cornea; *d*, a melanotic spot. (After Panas.)

The diagnosis is not usually difficult, particularly after ulceration has begun, but it is not always easy to tell whether the *pigmented patches* which are sometimes seen at the limbus are at present or prospectively malignant. A large number of the cases tabulated by Strouse originated in these black or brown patches, and in many cases the growth began to develop after a traumatism. Without doubt these patches are sometimes congenital and throughout life show no tendency to increase in size. On the other hand, sometimes they serve as the focus of a malignant process, particularly when they are not congenital. In the possibly malignant form the patch is black, and not brown as in the congenital variety, and it increases in size by a confluence with it of other patches or points which spring up in its vicinity.

Extirpation is the only treatment of malignant or supposed malignant growths. This should be done as early as possible and when the tumor is small, if a recurrence, which happens in a large majority of cases, is to be avoided. When it has attained any considerable size and its removal entails the loss of a large amount of tissue, it is better to sacrifice the eye at once by enucleation.

INJURIES TO THE CONJUNCTIVA.

On account of its exposed position, and in spite of the protection of the lids, the conjunctiva of the ball is a frequent subject of traumatic injury.

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One of the most common forms of injury is *burns* from chemicals or explosives. The most frequent and dangerous of these is that from *lime.* Unslaked lime, monoxide of calcium, coming in contact with the conjunctiva, absorbs rapidly the water of the tissues and gives out an enormous amount of heat, leading to a rapid destruction of the part. Seen immediately after the accident the conjunctiva looks white, as if seared with a red-hot iron. In most cases, unfortunately, the cornea is also involved. The accident is most commonly found among plasterers and those engaged in mixing mortar, and they have usually made matters worse by trying to wash out the material remaining in the eye with water. The burned tissue sloughs in due course of time, leaving a raw surface which heals by granulation. As both the bulbar and palpebral conjunctivæ are usually affected at the same time, there is great danger of union of the two opposing raw surfaces during healing, causing a *symblepharon.*

The immediate treatment of a burn from lime consists in the first place in the avoidance of all watery solutions. Sugar dissolved in water is recommended by some, since sugar forms an insoluble compound with lime, but it is likely to do as much harm as good, on account of the water it contains. An attempt should be made to saponify the lime that may be remaining in the eye by means of oil or fat of some kind,—lard being the form of fat usually most easily attainable at the time of such accidents. Milk is a good substitute. Usually, however, all the lime has been oxidized by the flow of tears long before the patient is seen by the surgeon. Still the oil treatment is the best that can be followed, as it affords a good protection to the burned surfaces. On account of its greater viscidity, castor oil is better than olive oil; it should be rendered aseptic by heat or by mixing it with boric acid. It must be continued through the entire course of sloughing and cicatrization, and cocaine and atropine can be mixed with it according to indications.

Careful search of the conjunctiva, and particularly of the cul-de-sac, should be made for remaining bits of lime or mortar. I have known an undiscovered small bit hidden in some granulation-tissue keep up a severe inflammation for weeks, which subsided immediately upon the removal of the foreign material.

The chief aim during the process of cicatrization should be to prevent a union between the opposing raw surfaces. For that purpose it has been proposed to wear a shield of glass, ivory, rubber, or other thin material between the lid and the ball. This is not only inconvenient, but also painful, and the same end can be attained by breaking up the adhesion twice a day by means of a probe carefully passed between the lid and the ball over the whole extent of burned surface back to the cul-de-sac. If the inflammatory reaction is at any time severe, it should be kept within bounds by cold compresses.

The fumes of ammonia are not only very irritating to the conjunctiva, producing hyperæmia, but when the action is sufficiently prolonged give

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rise to a pronounced conjunctivitis and even a destruction of the tissue. Cases have been reported (Trousseau, Abadie) in which the cornea was also seriously involved. It should not be forgotten that in all cases of burns of the conjunctiva by chemical substances the after-effects are apt to be progressive and may extend to the interior of the eye, causing cataract and other serious disturbances of nutrition.

Acids of various kinds may get into the eye and cause burns of the conjunctiva of greater or less severity according to the strength of the solution and its corrosive quality. The mineral acids, nitric, sulphuric, hydrochloric, etc., are the most serious. Washing the eye out with water, or with an alkaline solution if it is handy, is the appropriate immediate treatment. The subsequent treatment is the same as that for lime.

Burns from *carbolic acid*, even if the acid is pure, are not very serious in their consequences, since usually the epithelium only is affected.

Hot water, hot ashes, melted lead and iron, etc., sometimes find their way into the eye. The foreign substances should be removed as soon as possible after cocaine has been applied, and the treatment above outlined adopted or modified to suit the severity of the case. Burns from strong solutions of corrosive sublimate, nitrate of silver, etc., are to be treated on the same general principles.

Incised or lacerated wounds of the conjunctiva do not ordinarily require interference beyond aseptic dressings and bandage, except where they are extensive. It is then necessary to bring the edges of the wound together by means of fine sutures. There is little tendency to excessive suppuration in these cases if they are at once rendered aseptic.

FOREIGN BODIES IN THE CONJUNCTIVA.

Bits of cinder, coal, large particles of dust, etc., often find lodgement in the conjunctiva, particularly that of the lids. They give rise to great irritation sometimes when their presence is not suspected. In all cases of rather sudden pain and irritation of a single eye careful examination should be made for the presence of a foreign body. When found on the conjunctiva it can generally be wiped off with a bit of cotton wound on the end of a match. Only occasionally it is embedded so deeply as to require digging out with a needle or spud. The foreign substance which is most commonly found in the conjunctiva, however, is *powder*. In case of an explosion of powder in the face it is seldom that the conjunctiva escapes. The grains frequently pass through the conjunctiva and lodge in the sclera. They usually set up a considerable amount of inflammation if allowed to remain, which has the result of forming a small abscess around them, leading finally to their expulsion. In treatment it is usually better to pick out carefully, under cocaine, all the large grains, and douche the surface for a long time and frequently with a strong jet of aseptic liquid. This dissolves the powder grain and forces it out. In the course of time all will disappear, leaving, as a rule, no disfiguring mark. Very rarely the chestnut or other

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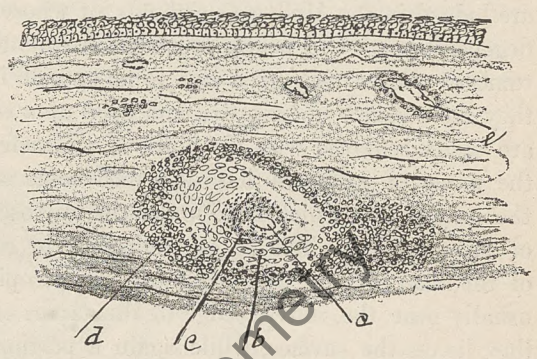
burr falls into the eye and leaves some of the spines sticking in the conjunctiva. On account of their light color and their semi-transparency, they are very difficult to find and remove. They should be carefully sought for, however, as they are more dangerous than other foreign bodies, on account of their tendency to wander into the adjoining tissues.

Sometimes even comparatively large bodies find their way into the retrotarsal folds, especially the upper one, and remain there without causing any very pronounced trouble for a long while, even years in some cases. "Crab's eye" or "eye stones" and flaxseed put into the eye for the purpose of chasing out foreign bodies are frequently found there long after their introduction has been forgotten.

The *hairs* of certain caterpillars (Bombyx pini, B. rubi) occasionally find their way into the conjunctiva and sometimes into the cornea as well, and give rise to a peculiar pathological condition known as *ophthalmia nodosa.* When it affects the conjunctiva it is characterized by great irritation and pronounced inflammation, with the development of small, firm, gray nodules in the substance of the conjunctival tissue. These nodules show a structure containing giant cells like tubercle and in the centre a hair of the insect. (Fig. 35.) The trouble is seldom limited to the conjunctiva, however; the cornea, ciliary body, iris, and chorioid are commonly involved in time, and very serious results may follow. These latter manifestations are due to the migration of the hairs. The pathology is somewhat obscure, but it is most probable that the inflammation is due to some chemical irritant in the hair, and that it is not simply a mechanical process. The appropriate treatment is the removal of the nodules as soon as they are discovered and combating the accompanying inflammatory symptoms. (For a further consideration of injuries to the conjunctiva, consult article on Injuries to the Eyeball.)

FIG. 35.



Ophthalmia nodosa (modified from Hanke): *a*, cross-section of hair; *b*, *c*, cells resembling giant cells; *d*, small round cells; *e*, section of blood-vessel.

COLORATION (STAINING) OF THE CONJUNCTIVA.

The most pronounced staining of the conjunctiva is that produced by nitrate of silver (argyriasis). This is always liable to happen when nitrate of silver is used long as a local application, and particularly when it is not immediately neutralized by chloride of sodium. The prolonged use of sulphate of iron has been known to leave a yellow coloration of the conjunc-

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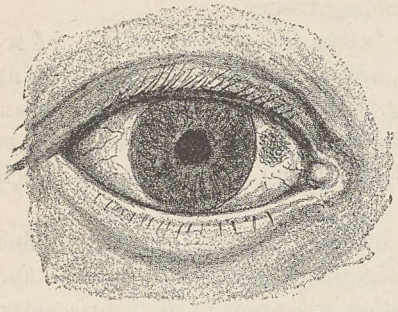
tiva,—*siderosis conjunctivæ.* The *brown spots* which are sometimes seen, especially on the conjunctiva of the negro, are due to deposits of pigment (Fig. 36), and are for the most part congenital, yet this brown coloration is much intensified in some diseases, notably in circumcorneal hypertrophy of the conjunctiva. A permanent black patch sometimes remains after dislocation of the iris under the conjunctiva.

Fig. 36.



Pigmentation of the conjunctiva.

Lithiasis of the Conjunctiva.—Small, whitish bodies are frequently seen buried in the conjunctiva of the inside of the lids, generally near the edge. They are hard, almost round, and usually extend above the general level of the conjunctival surface. They are calcareous formations, and are lodged in the Meibomian glands, of whose secretion they seem to be the degeneration. Old people are most frequently the subjects of these little tumors. Often they give rise to no special inconvenience, but sometimes they are a source of irritation and keep up a subacute conjunctivitis. They are easily removed with the point of a Graefe knife. Fuchs (1897) under the name of *pilzrasen* has described some small collections of fungi on the conjunctiva which have under the microscope somewhat the appearance of streptothrix. The spots, yellowish in color, vary in size from that of the head of a needle to almost microscopic magnitude, and are found usually near the convex edge of the tarsus of the upper lid. Sometimes they lie on the surface, while again a portion (supposed to be the mycelium) is sunk into the conjunctival tissue, pushing the epithelial layer before it. They resemble infarctions of the Meibomian glands, for which doubtless they have been mistaken. They are generally scraped off easily.

Leber (1895) has described a case of what he calls *conjunctivitis petrificans,* where the conjunctiva is the seat of an inflammatory swelling in which white opaque spots are to be seen that increase in size and finally coalesce, forming a mass as hard as stone. An examination of these concretions shows them to be a calcareous infiltration of the conjunctival tissue.

CONJUNCTIVITIS FROM INTENSE LIGHT.

Exposure of the eyes to an intensely strong electric light (Terrier) sometimes gives rise to a form of conjunctivitis distinguished by swelling of the lids, hyperæmia of the conjunctiva, and lacrymation, and attended with a good deal of pain. A large part of the painful symptoms accompanying *snow blindness* is referable to hyperæmia of the conjunctiva, and is due to the effect of the reflected heat of the sun on the exposed conjunctiva, making it a true *sunburn* of that membrane. The fine particles of snow driven into the conjunctiva also help towards this irritation. The phenomena are not

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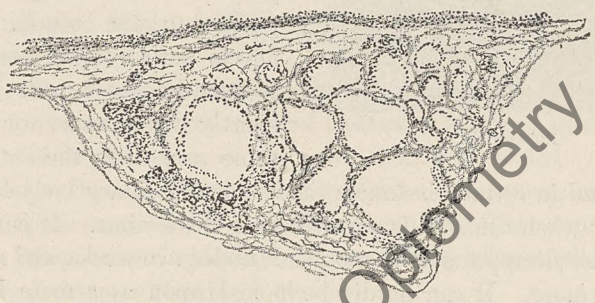
due, as was at one time supposed, wholly to the effect of strong light on the retina. The prophylactic treatment consists in the wearing of protective spectacles of blue or gray glass. Painting the skin underneath the eyes with a black pigment of some kind is also highly commended as a protection against the evil effects of reflection of sunlight from snow. The conjunctivitis is treated as is hyperæmia from any other cause, by boric acid solution with cocaine when required, cold lotions to the lids, abstinence from use, and protection from bright light.

AFFECTION OF THE CARUNCLE AND THE SEMILUNAR FOLD.

The caruncle, having essentially the same anatomical structure as the other parts of the conjunctiva, is subject to the same kinds of morbid processes. It possesses, however, some elements which other parts of the membrane do not,—namely, hairs, sebaceous glands, and other of the more truly dermoid structures; and these are occasionally the seat of pathological change. The hairs on the caruncle sometimes grow so long as to be a source of irritation, *angular trichiasis*, and to require removal. Abscesses also make their appearance here, and all the *benign tumors;* adenoma is probably the most frequent. Fig. 37 shows the histological structure of such

FIG. 37.



Adenoma of the caruncle, showing the glands lined with cells. (After Schirmer.)

a growth. A simple hypertrophy, the result of inflammation, is called *encanthis*. Sometimes a *calcareous deposit* is found in the substance of the caruncle. Malignant tumors of the kind already spoken of as occurring on the conjunctiva also occur here.

AFFECTIONS OF THE SCLERA.

The sclera, or protecting coat of the eyeball, having a smaller supply of blood-vessels and nerves, is not so liable to independent inflammatory troubles as the other parts of the globe, which are richer in those tissues.

It does not wholly escape, however, and there are two well-recognized forms of scleritis which are quite clearly defined in their clinical pictures. The first of these is—

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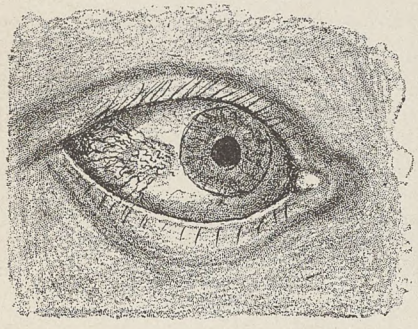
CIRCUMSCRIBED SCLERITIS, OR EPISCLERITIS.

In this affection there is a rather sharply limited elevation on the surface of the globe, usually some millimetres distant from the corneal edge, and generally to the temporal side. (Fig. 38.) It is reddish in color, with a violet tint at the centre which gradually fades off towards the periphery. It is not movable over the sclera, but seems attached to and to form a part of it, and the blood-vessels do not wholly empty themselves on pressure, as in the case of a purely conjunctival congestion. That there is also an exudation is evidenced by the elevation, sometimes considerable, of the spot above the surrounding surface.

The other portion of the conjunctiva shows little or no injection, and there is seldom any discharge of mucus or pus. Symptoms of irritation are, however, sometimes present in the form of easy and ready lacrymation and some photophobia. Pain is usually complained of, and frequently of a severe type. It is dull and aching in character, seldom neuralgic, and may be lacking altogether. Occasionally the spot is tender to the touch. It rarely affects both eyes at once, but quite frequently one attack is succeeded shortly by another or more. In some rare cases the cornea may be involved, and in certain instances the iris or the ciliary body is implicated, particularly when the spot is situated near the limbus. It is tedious in its course, seldom disappears, if pronounced, under two weeks, and may continue a month or more. It can hardly be looked upon as a mere local disease, but must be regarded as the manifestation in the eye of some systemic derangement. It has been considered by some as an expression of syphilis or gout, and probably the latter disease does sometimes manifest itself in this way. It is found, in this country at least, most frequently associated with the rheumatic diathesis.

Fig. 38.



Episcleritis.

The fibrous tissues, we know, are especially liable to be attacked by the rheumatic poison. Sometimes after the disappearance of the inflammation there remain darkish spots to mark the seat of the trouble.

The diagnosis is not difficult when the disease is once fully established. In the beginning it is liable to be confounded with a large phlyctenular elevation on the conjunctiva. In the latter disease, however, the centre is whitish yellow and the whole is movable over the sclera. Scleritis, moreover, is usually met with in adults, whereas the phlyctenulæ are much more common in childhood.

Schirmer (1895) found in cases of episcleritis an infiltration of numerous

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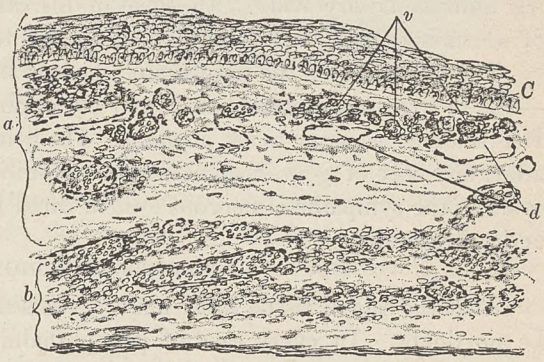
round cells with a fibrous-like exudation and dilatation of the lymphatics, especially in the subepithelial layer. (Fig. 39.)

Local treatment does not seem to have much influence on the course of the disease, in which particular it again resembles rheumatic inflammation in general. If there is much pain, palliative remedies, as cocaine or atropine, may be used, and hot applications, repeated three or four times a day, are always indicated. Dry heat is the best form. Excessive sweating by means of pilocarpine is commended by some as a hastener of resolution. Scarification of the mass has been recommended: the emptying of the blood-vessels may be of use at the beginning, but it can be of little or no benefit when the disease is once established. The subconjunctival injection of chloride of sodium solution or bichloride of mercury of weak strength, 1 to 1000, has been recommended lately by some good authorities.

FIG. 39.



Episcleritis: *a*, conjunctival and subconjunctival tissues; *b*, scleral tissue; *C*, conjunctival epithelium; *v*, blood-vessels; *d*, enlarged lymph-vessels. (After Schirmer.)

Even where the rheumatic or the gouty dyscrasia is not clearly pronounced it is wise to institute a proper general treatment of those affections, according as the one or the other is the more clearly indicated. Salicylate of sodium in large doses is often most beneficial. Of course, if there is even a well-grounded suspicion of syphilis, an antisyphilitic course should be followed. When there is photophobia, protecting glasses should be worn, and fatigue of the eyes should be avoided.

ACUTE HYPERÆMIA OF THE SCLERA (SCLERITIS PERIODICE FUGAX).

Under this head I place those occasional cases which have been observed in which there is a temporary acute hyperæmia of the conjunctiva and episcleral tissue coming on without any cause, acting directly on the parts, and disappearing in from a few hours to three or four days. The attacks are usually accompanied with pain, sometimes very severe, and generally with profuse lacrymation. The appearances resemble a beginning iritis, but an examination usually shows that the iris is entirely free from disease, as is also the cornea. Sometimes there is a slight œdema of the lids.

The peculiarity of the disease is its marked tendency to recurrence. Attacks may follow each other at intervals of a few weeks, though sometimes months elapse between them. Both eyes are never, or very rarely, attacked at the same time, but each may become affected alternately.

The pathology of the disease is by no means settled. Some cases

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described by Hutchinson (1884) under the name of "hot eye" in gouty subjects are probably of the same nature. Fuchs (1895) looks upon it as a true inflammation. I (1892) have attributed it to some transitory disturbance of the vaso-motor system. In one case under my observation, that of a lady about the climacteric, there were always premonitory symptoms of somnolency and a feeling of heat on the same side of the head as the eye which was attacked a day or two later. The true scleral tissue is never involved. It seems not improbable that some instances of temporary hyperæmia of this kind may be associated with a similar condition of the iris and the ciliary body. In some of his cases Fuchs observed a spasm of accommodation which was very likely due to this cause.

The treatment of the immediate attack is that of hyperæmia in general, —rest in a room moderately dark, and hot applications for the relief of pain. It is seldom necessary to use atropine. The general health should be looked after, any vice of gout, rheumatism, or syphilis attended to, and any disorder of the sympathetic nervous system cared for by proper mode of living and nerve tonics.

SCLERITIS ANTERIOR.

That the scleral tissue itself may be the seat of inflammation there can be no doubt. In every case of panophthalmitis it unquestionably takes part in the general inflammatory process, among other evidences of which is the great thickening of the scleral walls nearly always found in the stump extirpated after the inflammation has subsided.

The inflammation can be limited to a definite area and be confined wholly or mostly to the scleral tissue itself. This is observed more particularly at the anterior portion of the sclera and usually just over or near the ciliary region. We find here as the most marked feature of the process a thinning of the scleral wall, through which the dark uveal tissue is seen as a bluish spot. This usually becomes ectatic and forms a staphyloma of more or less magnitude. The development of this staphyloma is accompanied by pain, sometimes very severe, of a neuralgic character, lacrymation, and the usual inflammatory symptoms of a cyclitis or iritis. Some cases, however, are not so intense in their painful features, and the trouble is developed more slowly.

In some cases, too, the trouble begins at the margin of the cornea and extends to that tissue, making a sclero-keratitis. Very few cases of anterior scleritis occur without an implication of the ciliary body, and it has been a question whether the scleritis or the cyclitis were the primary disease. While unquestionably some cases begin in the ciliary body, as evidenced by the intense inflammatory symptoms for some time preceding the appearance of the staphyloma, there are others in which the blue spot appears after but little acute pain and much less inflammation. These staphylomata, which are the acme of the disease, are usually near the base of the cornea, and vary in size from two to five or six millimetres in diameter or sometimes even larger. There usually are more than one, and they

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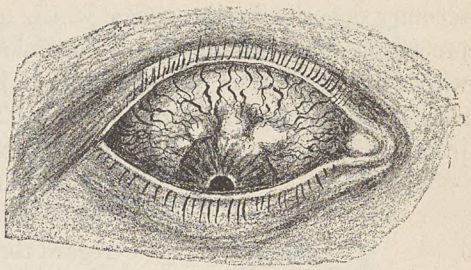
sometimes form a broken chain of nodules about the superior corneal base. (Fig. 40.) The disease has a tendency to recurrence, and sometimes after the subsidence of one nodule another appears at another place, thus showing its close alliance with episcleritis.

FIG. 40.



Scleritis anterior.

There is never any tendency to the formation of pus, and after a certain time, varying from three to six or eight weeks, the swelling subsides, leaving usually a bluish coloration to mark the seat. The sclera sometimes becomes very thin, and in one instance under my observation ruptured, giving issue to some vitreous. There is never a tendency to panophthalmitis, and, as a rule, the disease subsides without any marked deformation of the ball, though in those cases that I have seen there is always a permanent alteration in the curvature of the cornea, as shown by the ophthalmometer (astigmatismus acquisitus).

The only disease for which it is likely to be mistaken is the so-called gumma of the ciliary body. In this disease, however, the swelling at the corneal base is not bluish, but red and fleshy-looking, and follows a pronounced attack of iritis or cyclitis.

The principles of treatment are the same as those for acute inflammation of the ciliary body and the iris, one at least of which is nearly always involved. These comprise hot applications, rest, and atropine.

Undoubtedly most of these cases are the manifestations of a dyscrasia, and may be only the very advanced stages of episcleritis. In one case that I have seen, at least, there was a pronounced and obstinate rheumatic diathesis. It may also be one of the protean manifestations of gout or syphilis, and the possibility should always be held in mind in forming our therapeusis.

A species of *inflammatory thickening* at the posterior half of the sclera, in which the other portions of the eyeball did not participate, has been observed by Gayet (1888); and *partial hypertrophies* have been noted by Schnabel (1889), Todko (1874), and others. It is possible that some of these cases were instances of true *fibromata*.

Osteomatous degeneration of the scleral wall has also been reported.

Tuberculosis of the sclera is a rare independent affection. Müller (1890) reports one case.

Gumma of the sclera, independent of the ciliary body, has sometimes been seen. Andrews reports one case (1882).

Abscess of the sclera may occur either primarily (Leber) or as the result of trauma, or around a foreign body embedded in the tissue. It is to be

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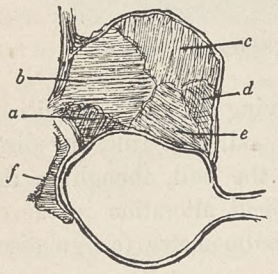
distinguished from an abscess of the conjunctiva by the fact that the conjunctival vessels are movable over it. The treatment consists in hot applications until fluctuation is evident, and its evacuation.

Melanotic Spots on the Sclera.—There are observed occasionally pigmentary deposits in the sclera which are not the results of pathological processes. They are more common in negro and other races and persons with a good supply of pigment than in blonds. The intensity varies from a light brown to an intense black. They are situated generally not far from the base of the cornea. But they may occur at other places, and there may be more than one on the same eye. Some authors (Hirschberg) are inclined to regard these spots as probable foci for malignant disease.

FIG. 41.



Melanosarcoma of sclera: *a*, *b*, *c*, *d*, *e*, lobules of the tumor, each having a different pigmentation and consistency, all enclosed in the conjunctiva; *f*, portion of the tumor covering the cornea. (After Addaris.)

Malignant Growths of the Sclera.—In an analysis of one hundred and thirty-seven malignant tumors of the exterior of the eyeball, Noyes (1879) found that twenty had their origin on the sclera. A much larger number (thirty-one) had their beginning at the sclero-corneal margin. Lying as it does between the conjunctiva and the chorioid, it participates in time in the malignant processes of each. It is very resistant, however, to invasion, and withstands infection for a long time. The tumors which originate in its own tissue are more likely to be sarcomatous or carcinomatous than epithelial. Fig. 41 represents a case reported by Addaris which seems to belong to this category. The same rule of treatment applies to these as to conjunctival tumors.

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DISEASES OF THE IRIS AND THE CILIARY BODY.

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THE CLINICAL EXAMINATION OF THE IRIS AND THE CILIARY BODY.

When examined by the naked eye, the anterior surface of the healthy iris shows some markings which it is important for clinical reasons to note attentively.

The circular pupil, lying approximately in the centre of the muscular diaphragm, forms, of course, the most conspicuous object. In health it is so freely mobile as to respond to every variation of light. This opening is braided, as it were, by a narrow line of black pigment formed by the pupillary border of the uvea. The band is more readily distinguished in a contracted than in a dilated pupil, and may be shortly termed the "uveal ring." Its width varies in different persons; for instance, it may be so narrow as to be identified only after careful examination, or, on the other hand, it may be broad and conspicuous. It is, however, invariably present, although often overlooked, in dark irides. It is thrown into striking relief by the white lens of cataract. The peripheral parts of the iris—that is, those that spring from the anterior portions of the ciliary processes—are hidden by the overlapping sclera, but in certain eyes some indication of their position may be noticed in the form of a darker, more pigmented area, running concentrically around the circumference of the iris. In this connection it may be of interest to recall the fact that, upon the average, 1.5 millimetres of the iris is hidden behind the apparent edge of the cornea. The membrane between the uveal ring and the ciliary margin is divided into two parts of unequal size by an elevated ridge termed the corona, corresponding to the small arterial circle of the iris, which lies beneath it. That section placed internal to the corona is called the pupillary zone, while that lying external is styled the ciliary zone. The former is distinguished from the latter not only by its relative narrowness, but also

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by its difference in pigmentation. Both zones are marked by delicate, lustrous, wavy ridges, interlacing with one another, and coursing radially from the periphery towards the centre of the iris. These ridges—always better developed in the ciliary than in the pupillary zone—are formed by the underlying vessels of the iris, and the angular gaps left between them are known as lacunæ or crypts. There is still another appearance to be noted: a close examination of the peripheral parts of the iris will show a number of grayish lines, forming segments of circles, and running concentrically with the margin of the cornea. They represent the folds into which the muscular screen is thrown during dilatation of the pupil, and are hence called "contraction furrows." Although varying in number and distinctness in different eyes, yet they are always more easily recognized at the outer and inner side of the iris than elsewhere, and with a contracted than with a dilated pupil. In many eyes spots of various colors may be seen upon the anterior surface of the iris. For the most part they lie in the external portions of the ciliary zone, and they are especially common in light-colored eyes. They are sometimes thought to present resemblances to various things, as words, initials, etc. In blonde subjects, moreover, a regular ring of whitish dots may be now and then observed.

To recapitulate: in the healthy iris the following markings may be usually distinguished: (1) the uveal ring; (2) the pupillary zone; (3) the corona; (4) the ciliary zone; and (5) the contraction furrows. A reference to the appended diagram will render these various points clear to the reader.

FIG. 1.

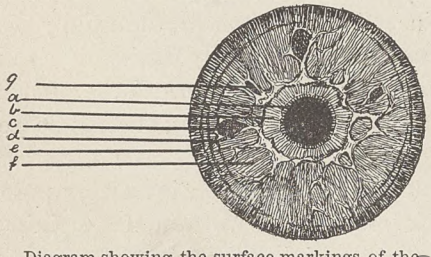

Diagram showing the surface-markings of the healthy iris: *a*, the uveal ring; *b*, the pupillary zone; *c*, the corona; *d*, the contraction furrows; *e*, external pigmented portion of ciliary zone; *f*, ciliary zone; *g*, a lacuna.

All the customary methods of clinical examination may at times be needed in order to detect affections of the iris and ciliary body. Nevertheless, it will be advisable to run over the more important in this place, as well as to indicate briefly certain special plans that are in use.

Simple inspection may afford a great deal of information. Thus, it will show whether the lustre and surface-markings of the iris are distinct, as in health, or blurred, as in inflammation; whether its hue is in any way modified or changed; whether nodules of lymph or vessels or hemorrhages are present upon its surface; whether its texture is obscured by any kind of change; whether it occupies its proper plane and position. Inspection will further tell us whether the pupil possesses its usual size, shape, position, and action; whether the anterior chamber and its contents are normal; whether there is any exudation into the pupillary area, upon the iris, or upon Descemet's membrane; and whether the cornea is transparent, as in health, or dimmed, as in some affections of the ciliary body. Then, again, it will show whether the

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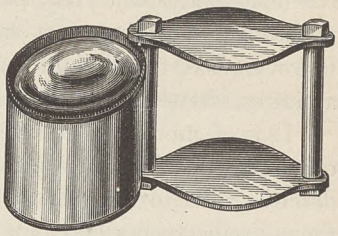
ciliary zone of the sclera possesses its normal appearance, or whether it is thinned or stained or bulging, as it sometimes is after recurrent irido-cyclitis and similar affections. Lastly, inspection will inform us whether the eyeball is unduly vascular, and, if so, what particular system of vessels is involved. Focal illumination (carried out in a darkened room) is often needed in order to render the finer changes visible, or to confirm the information already gathered in other ways, and a good deal of additional knowledge may be often gained by the use of a second convex lens to magnify the parts. The "corneal magnifier," or "loupe," shown in the figure, is a convenient instrument for this latter purpose. It is mounted in a metal frame, and, being equivalent to an inch and a half objective glass (50.0 D.), has considerable magnifying power; while, among its other advantages, it gives a flat field, free, or nearly free, from chromatic dispersion.

Fig. 2.



The corneal magnifier.

In health, as already pointed out, the anterior surface of the iris is beautifully reticulated, and shows a series of definite and characteristic markings. In some forms of disease, however, that is no longer the case. The exudative changes of iritis, for instance, damp those details to an extent that will naturally vary according to the type and severity of the inflammation. In slight cases the iris may be merely "muddy," but in more marked ones it may be obscured by exudations of lymph, which sometimes show a curious reticular or "spider-web" appearance. Again, the lustre of the iris may be dimmed by inflammation. That character depends upon the structural integrity and well-being of the endothelial coating, which is practically always altered in iritis. Hence a lack-lustre look of the iris is often an important sign of slight and early disease.

Everybody is aware that the iris may vary in color, not only in different persons, but also in its different segments in the eye of one and the same individual. Its hue depends upon two factors, the one practically invariable, the other variable. The first, or invariable, factor is constituted by the layer of pigmented epithelial cells that lines its posterior surface, the *pars retinalis iridis* of anatomists. This is present at birth, so that the eyes of babies appear by interference to be of a dark slaty-blue color (Aristotle). With advancing years, however, the cells of the stroma of the iris become more or less pigmented, and the ultimate color will depend upon the relative proportion that this bears to the epithelial pigmentation. For example, a large amount of surface pigment will impart to the iris a dark shade, while a small amount, on the other hand, will cause it to appear gray or even blue. As a rule to which exceptions are both numerous and important, blondes will have a light and brunettes a dark-hued iris; which is equal to saying that a certain relation exists between the pigmentation of

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the cutaneous structures and that of the iris. The colors that are usual in this country, it may be noted in passing, are gray, blue, hazel, and brown.

Instances of *heterochromia—i.e.*, marked pigmentary differences in the two eyes of the same individual—are now and then encountered, but a more common condition is that in which a sector of one iris possesses one color, while the remaining part has another. These physiological anomalies, however, need not detain us in this place, inasmuch as they have received attention elsewhere. (See volume i., page 434.)

It is of more importance to note that the existence of inflammation is often revealed by subtle changes in the color of the iris more difficult to describe than to recognize. It may be broadly said, however, that under these circumstances blue or gray gives way to green, while brown yields to various shades of red. The alteration—which is usually better marked in the pupillary zone than elsewhere—may be general or local in its distribution. For example, in some cases of cyclitis, the iris, as a whole, has a peculiar grass-green tint that, once seen, can be scarcely forgotten; on the other hand, the change may be limited to that particular portion of the iris which forms the chief focus of inflammatory action, as shown by certain cases of traumatic iritis. Color-changes, although easy to appreciate when one eye is alone involved, have less diagnostic importance when the ailment is bilateral. Indeed, it may be laid down almost as a rule that iritis or irido-cyclitis is present in every instance where the iris of an inflamed eye differs markedly in color from that of its fellow. But, in attempting to estimate color, the surgeon must never neglect to pay particular attention to the condition of the cornea and the aqueous humor; for if those media be cloudy, the apparent may be very different from the real color of the iris.

In the normal condition of things the iris, as seen from the front, has a slightly sloping surface; or, in other words, lies deeper at its periphery than elsewhere. Seen sideways, it takes, in fact, the shape of a blunt cone, of which the truncated extremity is formed by the pupil, the base by its attachment to the ciliary body. This means, of course, that it is supported by a portion of the anterior surface of the crystalline lens, and to some extent is moulded upon the contour of that body. Hence, when the lens is absent, the plane of the iris hangs vertically, and, having lost its support, manifests a peculiar tremor (*iridodonesis*) whenever the eye is quickly moved. It must not be forgotten, however, that when the pupil is greatly contracted a slight quivering of the iris is now and then observed in perfectly normal eyes.

The plane of the iris and, consequently, the form of the anterior chamber may undergo considerable alteration in some affections of the uveal tract. A very curious state of things is sometimes observed in cyclitis, the chamber being now shallow, now deep, the changes taking place during the course of a few hours. These remarkable alterations are associated with corresponding fluctuations of tension. When the pupil is

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"excluded," again, the iris, bound down by its pupillary and ciliary attachments, may be bellied forward at its intermediate parts, like a sail full of wind, so that the anterior chamber is obliterated, save at its central and peripheral parts, which by contrast may even appear to be deepened. This condition, often spoken of as *iris bombé*, is a pathognomonic sign of secondary glaucoma. Then, severe cases of irido-cyclitis are generally associated with inflammatory exudation posterior to the iris, which eventually entails deepening of the anterior chamber, a change especially marked at its periphery ("*retraction of the iris*").

The contents of the anterior chamber, also, may be modified in various ways. Thus, the aqueous may be rendered uniformly turbid by the admixture of products thrown off from an inflamed iris or ciliary body; deposits of blood or of pus may be present; while very rarely a spongy or gelatinous exudation may be observed in the anterior chamber. Foreign bodies, such as eyelashes, may occasionally be seen free in the aqueous humor, a remark that applies also to tumors and to parasites, such as the cysticercus or the filaria sanguinis.

The clinical examination of the pupil is so important as to merit more than a mere passing mention. Its shape, its position, its size, and its reaction to light and to accommodation will have to be investigated. In many affections, both of the eye and of the nervous system, it may be modified in any or in all of these respects.

The patient is placed opposite a window in such a way that the light falls equally upon both his eyes. The first step will be to ascertain whether the pupils occupy their natural position and possess their normal shape. Contrary to current notions, the pupil does not occupy the precise centre of the iris, but nearly always lies somewhat to the nasal side of that point. In some instances, indeed, it is markedly eccentric, and in the congenital anomaly known as *corectopia* it may even be placed close to the margin of the cornea.

When synechiæ are present, the shape of the pupil may be altered in various ways. It may, for instance, be alternately toothed and notched, or scalloped, as it were. It may be oval, stellate, triangular, quadrangular, hourglass- or kidney-shaped, or it may be altogether irregular in outline. A striking conformation, depicted in the accompanying diagram (Fig. 3, No. 4), has been aptly likened to the ace of clubs. The synechiæ themselves may be wholly or partly pigmented. They may be long or short, thin or thick, many or few. They may or may not be connected with deposits of lymph upon the anterior capsule of the crystalline lens (Fig. 3, No. 5). There is a curious kind of adhesion which is produced by the precipitation of fibrinous deposits from the aqueous humor. These may lodge at the margin of the pupil, which may in that way become tagged down to the anterior capsule of the lens. In this event the synechia, instead of arising, as in the common form, from the uveal layer, springs from the front of the iris. Anterior synechiæ also may be brought about in a similar way.

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As to abnormal movements, the rapidly alternating contractions and dilatations known as hippus may be very occasionally observed. The condition, as pointed out by Gunn, sometimes marks the point at which sympathetic neurosis is about to yield to sympathetic inflammation, but in the majority of cases its cause or causes remain obscure.

The next step will be to measure the size of the pupil. In this connection, however, it must be remembered that there is no uniform standard, or, in other words, that the pupil varies in size according to the individual. The average diameter has been given by Jaeger and by Merkel as 4 milli-

FIG. 3.

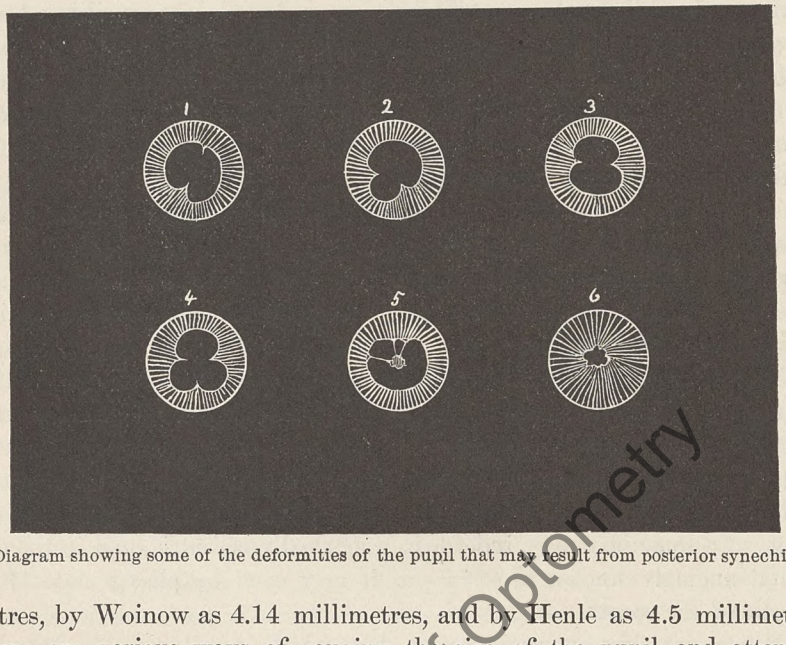

Diagram showing some of the deformities of the pupil that may result from posterior synechia.

metres, by Woinow as 4.14 millimetres, and by Henle as 4.5 millimetres. There are various ways of gauging the size of the pupil, and attention may be now drawn to some of the more useful.

The simplest plan is to direct the patient to fix a distant object, the surgeon meanwhile holding a millimetre rule as close to the cornea as possible. The transverse measurement of the pupil is then read off from the scale. This rough-and-ready method cannot be regarded as wholly satisfactory, and in practice it is often replaced by the use of special instruments (pupillometers), of which there are several in common use. A well-known pattern is that bearing the name of Edgar Browne. It consists of an ivory plate pierced by a series of circular holes that range in diameter from .5 millimetre to 7.5 millimetres. The little instrument is placed close to the patient's eye, and the size of the pupil is estimated by means of the guide-lines shown in the illustration. In another form, circular disks of known size are painted upon the pupillometer, and the diameter of the pupil is determined by a process of simple comparison.

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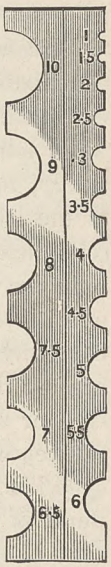
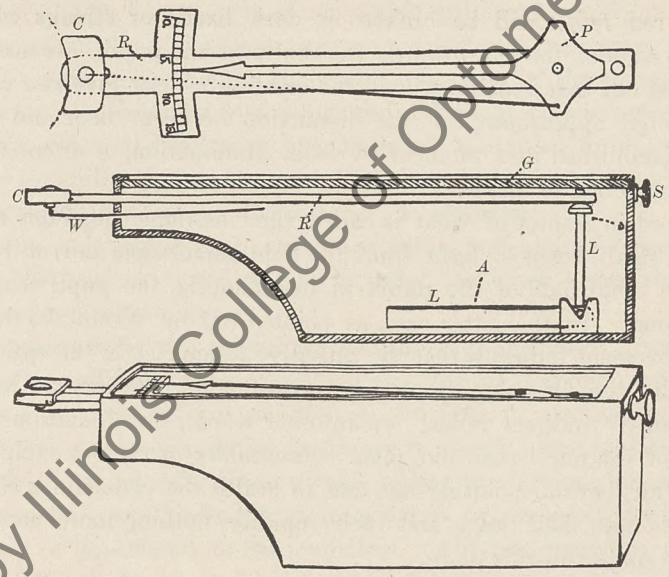
Randall's instrument consists of a metallic disk pierced by a number of circular holes ranging in diameter from one millimetre to ten millimetres. In use, it is held in front of the patient's eye and rotated until an aperture is found that corresponds with the size of the pupil. The size and shape of Jessop's pupillometer are shown in the appended illustration. Its chief advantages appear to be: (1) that the dimensions of the pupil may be quickly ascertained, and (2) that the semicircular apertures can be placed in front of the iris without at the same time cutting off so much light as to vitiate the measurement by dilatation of the pupil. Another instrument often employed at the present time takes the form of a metallic disk, marked by black circles of known size, and constructed in such a way that it may be fixed upon the ophthalmoscope instead of the ordinary revolving plate that drives the lenses. The contrivance is known in this country as Morton's.

FIG. 4.



Priestley Smith's tonometer, which depends upon a different principle, may be also used to measure the size of the pupil. It consists of two plano-convex lenses cemented together and enclosing a millimetre scale. The instrument is held as close as possible to the patient's eye and at its focal distance (ten inches) from that of the surgeon, and the transverse diameter is then read off from the millimetre scale. The diagram, with its appended explanation, will render the working of the tonometer clear to readers.

FIG. 5.



The upper figure shows the ram *R*, carrying the crescent *C* and the pointer *P*, these two being connected by a wire,—seen from above. The second figure shows the instrument in section, the end of the ram resting on the upright arm of the rectangular lever *L*.

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In order to test the reaction of the pupil to light,—the "direct reflex," as it is termed,—one eye is completely excluded, while the gaze of the other is directed towards a distant object. The open eye is then shaded by the surgeon's extended hand, when its pupil will become considerably larger, returning, after a few slight oscillations, to its original size when the hand is removed.

Inconspicuous posterior synechiæ often become visible during this examination, which is, indeed, the ordinary clinical method of looking for them. Theoretically, the pupil should remain motionless to light when adhesions are present at every part of the pupillary edge. Practically, however, that is by no means always the case, since the stroma of the iris appears to be capable of gliding over the layer of pigmented cells that lies behind it. Hence some alteration in the pupil under the influence of light does not exclude circular posterior synechiæ. When the uveal layer is extensively tied down to the anterior capsule of the lens, it is nevertheless a fact that the iris, as a rule, fails to react to light, probably because the tissue has undergone serious structural alteration. The presence or absence of pupillary movements, therefore, constitutes a diagnostic difference between mere "exclusion," on the one hand, and total posterior synechiæ, on the other. Lastly, it should be mentioned that a sluggish pupil is not infrequent in the early stages of iritis, a sign easy to appreciate if one eye be alone inflamed.

In addition to the method just described, there is another way of detecting the presence of synechiæ, namely, by the "mirror test." This may be described as follows: the light from an ordinary concave ophthalmoscopic mirror is thrown into the eye, when, if adhesions be present, the circular red reflex will be broken by dark bands or streaks which run from the edge of the iris towards the centre of the pupil. It need hardly be pointed out that opaque striæ in the crystalline lens give rise to a somewhat similar appearance, but the distinction between them and synechiæ may be established in a moment by focal illumination, or often enough by simple inspection of the eye.

A word in respect of what is called the "*hemiopic pupillary reaction.*" When a small beam of light from the ophthalmoscopic mirror is thrown upon the blind half of the retina in hemianopsia, the pupil may or may not respond. In the latter case, as pointed out by Wernicke, the loss of reflex movement indicates that the causative lesion lies in the optic tract.

Besides the foregoing, it may be occasionally necessary to investigate the so-called "indirect reflex," or, in other words, the dilatation and contraction of the pupil that take place consensually in the eye excluded from light. But, beyond pointing out that in health the two actions correspond as regards both their range and their rapidity, nothing more need be said upon the subject in this place.

The main points, then, to be attended to in testing the pupillary reflexes may be thus summed up: 1, to examine each eye separately; 2, to take care

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that the eye not under examination is wholly excluded from light; 3, to cause the patient to "fix" an object lying at least twenty feet from him.

It is customary, as already implied, to carry out the tests detailed above in ordinary diffused daylight, and this suffices in the majority of cases. Sometimes, however, it is advisable to conduct the examination under artificial light. Thus, the surgeon may be uncertain in some cases whether the pupil reacts at all, under which circumstances the point may be settled by concentrating the light from an Argand burner upon the eye by means of a convex lens, and using the loupe to magnify the parts. This method of examination, too, has its special value in recognizing the fine synechiæ or pigment dots now and then met with in iritis; in observing the dilated vessels associated with cyclitis; in identifying delicate filaments of persistent pupillary membrane; or in finding the minute lacerations of the free border of the iris often, if not always, associated with so-called traumatic mydriasis.

The pupil, as everybody knows, contracts during convergence and accommodation. This "associated action," as it is called, is tested clinically by getting the patient to "fix" a distant object, the surgeon meanwhile noting the size of the pupil. The patient's gaze is next directed towards any convenient article—as, for example, the top of a pencil-case—held from twelve to fifteen millimetres from his eye. The size of the pupil being noted under the latter conditions and compared with the measurement formerly obtained, the surgeon will be in a position to form an estimate both as to the range and as to the extent of the associated action.

A simple clinical way of carrying out this test is for the patient to look at a distant object through the interval between the surgeon's outspread index and middle fingers, held about twelve millimetres in front of the eye under examination. The fingers are next closed, and the patient is directed to fix his gaze upon them. In certain affections of the nervous system the reflex action may be lost, while the associated action remains intact. The term "Argyll-Robertson phenomenon" is applied to this condition, which is generally regarded as symptomatic of tabes dorsalis, and is said to occur in eighty per cent. of the cases (F. T. Roberts).

Finally, it is important to record the nature and kind of light employed during these various examinations, in order that such knowledge may be available for future reference.

One cannot leave this subject without mentioning that in suspected iritis the use of a mydriatic is sometimes necessary for the purposes of an accurate diagnosis. A one per cent. solution of sulphate of atropine is generally selected, and a drop or more of the liquid is placed in the conjunctival sac. In health the pupil will dilate within twenty-five minutes or so after the application has been made, but an inflamed or hyperæmic iris will respond sluggishly or imperfectly to the mydriatic. Any iris, therefore, that reacts fully and equally to a mydriatic within the time named may be safely assumed to be free from inflammation. If isolated synechiæ be present,

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the iris will retract between the adhesions, which naturally form so many fixed points binding it to the capsule of the lens (see Fig. 3). In this way the pupil will manifest those changes in outline alluded to on an earlier page. The use of atropine, moreover, will tell us whether the pupillary edge of the iris is wholly tied down by inflammatory deposit to the lens-capsule ("exclusion"), or whether it still remains free at some part of its circumference, in which event small indentations will of course be noticed at one or more points of its pupillary edge.

Whenever practicable, the eye should be examined with the ophthalmoscope, inasmuch as valuable information may in that way be now and then obtained. Simple iritis is not associated with changes in the vitreous, whereas the inflammatory deposits of cyclitis not infrequently pass into this humor. Under such circumstances they may be recognized as fixed or floating opacities, varying in size from particles as fine as dust to exudations of large dimensions. Although focal illumination may suffice to identify them in some instances, yet, as a rule, direct examination with the ophthalmoscope is needed. Atrophy of the iris, as will be explained later, may follow long-continued irido-cyclitis, intra-uterine iritis, or chronic glaucoma. Under these circumstances the attenuated tissues allow light to pass through them at one or more points, a change that is most easily appreciated during ophthalmoscopic investigation. Furthermore, the choroid, the retina, or the optic disk may participate in affections of the ciliary body, so that ophthalmoscopic examination may in that event be of great service to the surgeon in making a prognosis.

Attempts have been made to examine the ciliary region of the fundus by means of a strong achromatic prism combined with a weak spherical lens (Galezowski, Schwartzschild.) After dilatation of the pupil, this little apparatus is held at its focal distance from the eye, opposite the point to be investigated. A beam of light is then thrown into the eye from an ophthalmoscopic mirror, as in the ordinary examination by the indirect method, the convex lens being replaced, however, by the sphero-prism. Galezowski claims that by this plan characteristic changes may be recognized in the region of the ora serrata in specific iritis, interstitial keratitis, etc.

A special method has been employed to distinguish between a simple staphyloma and one caused by an underlying tumor of the ciliary body. The "light-test," as it may be called, is thus carried out. The surgeon stations himself in front of the patient and fixes his attention upon the pupil. An assistant then concentrates a powerful beam of light upon the ciliary protrusion. It is asserted that the light cannot penetrate into the globe if the swelling be caused by a new growth, while the beam will be recognized through the pupil should it be merely in the nature of a staphyloma.

An investigation of the form-sense has an important bearing upon diseases of the iris and ciliary body, and so should not be omitted. Thus, pure iritis is seldom associated with much defect in sight, unless the aqueous be clouded or the pupillary area blocked by inflammatory material, or

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the posterior surface of the cornea be sprinkled with deposits, under which circumstances the reduction is proportionate to the degree of obstruction. On the other hand, marked deterioration of vision is a common feature in cases of cyclitis, and is due to inflammatory deposits behind the iris or to changes in the vitreous humor. It is obvious, therefore, that the condition of the sight forms an important factor in distinguishing between these two maladies, and may, moreover, tell us when one is passing into the other.

Whenever possible, the refraction of the eye should be ascertained, inasmuch as no trustworthy conclusion as to sight can be reached unless that be done.

It is important to note that temporary alterations in refraction are by no means uncommon, both in iritis and in so-called serous cyclitis. They usually take the form of myopia, which makes its appearance during the attack and disappears after its subsidence. Schapringer[1] has suggested that these alterations are due to a temporary increase in the refractive index of the aqueous humor, which he supposes to be brought about by an excess of fibrin. Oliver has shown, however, that in most cases the myopia appears to depend upon spasm of the ciliary muscle.[2]

The tension of the eyeball also should always be investigated. In fact, its estimation is one of the most valuable means of diagnosis and prognosis possessed by the surgeon. Variations of tension are indicative of cyclitis, but it should be carefully noted that in iritis, on the contrary, such changes are not met with, at any rate in the common run of cases. Some variation of tension, therefore, may give the surgeon the first hint that an inflammation hitherto confined to the iris has invaded the ciliary body. Again, in the later stages of cyclitis a persistent diminution in tension may indicate that the eyeball is on the high-road to atrophy, or *phthisis bulbi*. When the pupil is "excluded," or when the iris or the ciliary body is the seat of a new growth, undue hardness of the eyeball will warn us that secondary glaucoma has set in.

Tenderness of the globe constitutes a characteristic feature in many cases of cyclitis, and may even serve to distinguish that condition from iritis. Its presence is often elicited during the estimation of tension, but it may be specially sought for by gentle pressure made upon various parts of the ciliary region with the finger or the probe through the closed lids. The tenderness may be confined to one particular area, or it may, on the contrary, have a more general distribution. Where one eye is alone affected, the sound eye should be taken as a standard for comparison.

Lastly, the surgeon should note the condition of the eye with regard to vascularity, since that not only forms a trustworthy guide as to the severity of any inflammation that may be present, but possesses as well a

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[1] New York Medical Journal, October 21, 1894.

[2] Oliver, C. A., Transactions of the American Ophthalmological Society, 1892 and 1894.

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diagnostic value. In acute iritis the cornea will be encircled by a pinkish annulus, which, upon careful examination with the magnifying glass, resolves itself into a series of fine, closely set, parallel twigs, derived chiefly from the episcleral branches of the anterior ciliary arteries. In typical form, the cornea, then, is surrounded by a vascular ring varying in width from two millimetres to five millimetres, while the rest of the eyeball retains its natural color. For the sake of brevity, the condition is commonly spoken of as *ciliary* or *circumcorneal* congestion. In cyclitis the congestion has much the same general character, although it often manifests a tendency to affect certain portions of the ciliary region in preference to others, and is, moreover, violet rather than pink in hue. The vessels of the conjunctiva, too, often sympathize in iritis and cyclitis, and the extent to which they are involved will be roughly proportionate to the intensity of the inflammation. The two forms of vascularity may be readily distinguished both by the different arrangement and color of the component vessels and by the fact that the conjunctival twigs may be moved along with the membrane in which they lie. Again, the latter may be easily emptied of their contents by pressure, which is not the case with the ciliary branches. Clinically, this point is ascertained by pressing the lower lid against the inferior sclero-corneal junction, and afterwards sliding the lid downward over the surface of the eyeball. If the conjunctival system be alone engorged, the pressure-track will remain white for a brief moment; if the ciliary vessels be also involved, a pinkish area continuous with the corneal margin will remain unaltered by the manipulation. Lastly, ciliary congestion has its point of greatest intensity around the cornea, while conjunctival congestion is most marked towards the culs-de-sac, superior and inferior.

The type of congestion changes when the tension of the eyeball is heightened, as it may be in some of the affections dealt with in this article. When that is the case, large, tortuous vessels, which emerge from the globe at some little distance from the margin of the cornea, may be noticed upon raising the upper lid. They course beneath the conjunctiva, and are formed by the anterior ciliary veins.

INFLAMMATION OF THE IRIS AND OF THE CILIARY BODY.

PRELIMINARY REMARKS.

The iris and the ciliary body together form the anterior division of the middle tunic of the eye,—that is, of the uveal tract. They are anatomically continuous; are supplied in common with arterial blood by the long posterior and anterior ciliary vessels; and present many points of resemblance in their histological structure. They are often involved, although not necessarily to an equal degree, by the same inflammatory process, which in many cases seems to strike both structures simultaneously. Apart from that, it may be broadly stated that most inflammations arising in the ciliary

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body will sooner or later pass over into the tissues of the iris, and thus give rise to the mixed form, irido-cyclitis: to vary the statement, cyclitis is rare without iritis. An inflammation primarily attacking the iris may, however, remain limited to that membrane through its entire course. At the same time, iritis is not infrequently complicated with cyclitis, although passage of inflammation from iris to ciliary body is distinctly less frequent than the reverse process.

The fact that iritis and cyclitis so often form part and parcel of one and the same affection has important clinical bearings, and may be explained in the following simple way. As already pointed out, the two structures are in intimate connection, both anatomically and by the possession of a common blood-supply. Hence there can be little obstacle to the passage of the textural changes of inflammation by the former and of the vascular changes by the latter path. It is not surprising that inflammation of the ciliary body is very generally transmitted to the iris, since almost all the arterial blood received by the last-named structure has to pass in the first instance through the ciliary body. On the other hand, it is conceivable that transmission from iris to ciliary body would be attended by greater difficulties, inasmuch as the vascular processes would have to travel, as it were, against the blood-stream. In short, it is possible that while continuity of tissue and vascular supply unite in transmitting inflammation from ciliary body to iris, the former means comes chiefly into play when the ciliary body is affected secondarily to the iris. Moreover, the aqueous humor bathes both structures. It has been suggested that changes in the chemical composition of the fluid may possess some influence in spreading disease from one to the other. Mechanically, at all events, we know this to be the case: the cellular deposits thrown off from an inflamed ciliary body often pass into the aqueous humor and are precipitated into the pupillary area or projected against the posterior surface of the cornea, where they appear to have the power of setting up further mischief.[1] Lastly, it must not be forgotten that the causes of iritis are, with scarcely an exception, those of cyclitis also, a fact that may well explain the common clinical association of the two disorders.

From what has been said, it must be obvious, then, that although for the sake of convenience iritis and cyclitis will be separately described in this communication, the signs and symptoms of the two maladies are in actual practice often combined.

The uveal tract, as we know, nourishes some of the most important parts of the eye, for example, the crystalline lens and the vitreous humor. It is the source, moreover, of the aqueous humor. As a consequence of this, the nutrition of those structures frequently suffers in inflammatory

[1] Alt finds that an exudation in the anterior chamber often stimulates the endothelial lining to rapid karyokinesis, so that clusters of round, vesicular cells soon form upon the original layer. These often seem to coalesce and to form polynucleated giant cells. (American Journal of Ophthalmology, February, 1896.)

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affections of the iris and ciliary body. It must be borne in mind also that the tissues of the choroid run into the ciliary body at the ora serrata. Hence the former now and again participates in affections of the latter.

HYPERÆMIA OF THE IRIS.

By many surgeons a clinical distinction is drawn between hyperæmia and inflammation of the iris. The former is not infrequently observed after injuries to the eye, such as may be inflicted by blows or by the presence of foreign bodies. It is also met with in the course of corneal ulcers, particularly if they manifest a tendency to rapid spread; after the application of jequirity; and in many other conditions that need not be more particularly specified.

Hyperæmia is marked by a contracted pupil, which reacts sluggishly both to light and to mydriatics. Slight, almost inappreciable, discoloration of the iris is very generally present, a sign that may be limited to a particular part of its surface or may have a more general distribution. The remaining symptoms include a trivial ciliary blush (that may, like discoloration, be partial or general), together with a variable amount of lacrymation and photophobia. Hyperæmia, provided it does not form the initial stage of inflammation, speedily subsides upon removal of its cause, and entails no bad results.

SYMPTOMATOLOGY OF IRITIS.

The chief signs and symptoms that attend actual inflammation of the iris may be thus classified:

1. Pain, photophobia, lacrymation, etc. 2. Redness of the eye. 3. Impaired mobility of the iris. 4. Exudation of inflammatory products. 5. Disturbance of sight, etc.

1. Pain, Photophobia, Lacrymation, etc.—Some amount of pain commonly accompanies inflammation of the iris, but the surgeon must be prepared to recognize the clinical fact that its intensity varies much. Thus, in the insidious form spoken of as "quiet iritis" it is generally absent altogether; while in the so-called "neuralgic" variety it constitutes one of the most marked and troublesome symptoms. It may be variously described as dull or sharp, as aching or throbbing, as cutting or stabbing. Always more or less paroxysmal, it is generally worse at night than at other times. Patients not infrequently remark that it is heightened by movement of the eyes. It has a special tendency in some forms of iritis to overflow, as it were, from the eyeball into the brow, temple, side of the nose, cheek, and gums; to radiate, in fact, into those parts supplied by the supra-orbital and infra-orbital branches of the fifth cranial nerve. In these circumstances the rim of the orbit and neighboring structures are not infrequently found to be painful upon pressure.

Closely allied to pain stand three other signs, namely, general irritability of the eye, lacrymation, and photophobia. As a rule, these bear some relation to the amount of pain, being more marked when that is

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severe, and *vice versâ*, although they may attain a high grade in the absence, more or less complete, of that symptom. Dread of light (a very variable symptom) is never so intense in iritis as in some forms of keratitis. Lastly, it may be noted that spasm of the orbicularis muscle is practically sure to be met with when photophobia is marked.

A certain amount of constitutional disturbance may be met with, but only when the foregoing conditions are present in aggravated form. This is especially likely to be the case in neurotic subjects, as well as in those whose bodily state has been undermined by former excesses. In cases of this kind the temperature may be raised one or two degrees, the skin may be dry, the tongue coated, the urine thick, the bowels constipated, and vomiting may take place; in a word, some amount of fever may be present.

2. **Redness of the Eye.**—As already stated, redness of the eye is present at some point in the history of almost every case of iritis. It is, perhaps, the first sign to appear, while it is certainly one of the last to disappear completely. It possesses, therefore, considerable practical interest. The classical condition is for the cornea to be surrounded by a pink belt mainly composed of the distended episcleral twigs of the anterior ciliary arteries. The redness is greatest close to the cornea, and shades away towards the equator of the globe. The width of the colored area may vary according to circumstances. Thus, in mild cases it may be insignificant, but in severe ones it may have a breadth of five millimetres or so. The zone may be complete or partial in its distribution, corresponding in the latter event with a partial iritis. In some cases a narrow ashen-gray line (formerly believed to be characteristic of rheumatic iritis) may be observed between the apparent edge of the cornea and the encircling redness. This "ciliary" or "circumcorneal" redness, it is important to note, is by no means absolutely diagnostic of iritis, inasmuch as a somewhat similar appearance is met with both in keratitis and in cyclitis, although in the last-named affection the congestion, as will be explained later, is more commonly violet than red in hue. A diagnosis, therefore, must never be based exclusively upon its existence. It must be added, too, that the conjunctival vessels are generally involved in severe iritis, and when this is the case the underlying ciliary system will be more or less hidden. Under such circumstances some chemosis, together with swelling of the upper lid, is usually present.

As the ways of distinguishing between ciliary and conjunctival congestion have been described upon an earlier page (page 266), they need not be recapitulated.

3. **Impaired Mobility of the Iris.**—An imperfect action of the pupil, both to light and to mydriatics, constitutes one of the earliest and most characteristic signs of iritis. At a later stage there may, indeed, be no pupillary movement whatever, and that quite apart from mechanical obstacles to dilatation, as posterior synechiæ. The cause of this partial or complete immobility must almost certainly be sought in a tonic contrac-

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tion of the sphincter muscle, the result of irritation of the ciliary nerves that is undoubtedly present in most cases. The pupil, too, is always more or less contracted, a point easy to appreciate if the ailment be one-sided. It seems likely that the contraction is mainly caused by a general engorgement of the vessels of the iris, although it stands to reason that some share must be also taken in this respect by spasm of the sphincter iridis.

4. **Exudation of Inflammatory Products.**—Every case of iritis is accompanied by more or less exudation of serous, plastic, or purulent material. A little consideration will show that this may occur in any or all of the following directions,—viz., (*a*) into the tissue of the iris; (*b*) upon its anterior or its posterior surface; (*c*) into the pupillary area,—*i.e.*, that part of the anterior capsule of the lens left exposed by the pupil; (*d*) into the aqueous humor.

(*a*) The first hint of its presence is afforded by alterations in the texture of the iris, which becomes dull, muddy, thickened, and felted to an extent that will vary according to circumstances. If inflammation be of slight intensity, for instance, the iris, wholly or in part, may appear somewhat swollen, while its surface-markings may be, perhaps, rather indistinct. In more severe cases, on the other hand, it becomes obviously thickened, a change that affects its ciliary as well as its pupillary zone. In that event, the delicate, wavy, interlacing ridges normally present are more or less concealed, the crypts also being indistinguishable. A further appearance, now and then noted, is the existence of narrow, tortuous, red lines, which represent the vessels of the iris gorged with blood and pressed forward by exudation behind them. These are generally seen in the pupillary zone only, and when present are occasionally associated with minute extravasations of blood.

(*b*) The earliest sign of exudation upon the anterior surface of the iris is shown by some alteration in its epithelial covering, which looks as though it had been breathed upon, and appears slightly roughened; in other words, there is an absence of the lustre normally present. Changes in color are next observed, which in mild cases remain limited to the pupillary zone, or even to particular portions of the latter. It is difficult to define accurately the nature of these alterations, but they tend, as a rule, to various shades of red and green, the exact hue depending almost wholly upon the natural tint of the iris. Round or oval nodules of rusty color are very generally, if not always, present in the iritis of secondary syphilis, and may be regarded as pathognomonic of the affection in question. They seldom exceed two millimetres in diameter; and, although they may be met with in any part of the iris, they nevertheless affect by preference its pupillary edge,—that is to say, a situation where the blood-supply is abundant. Individual "condylomata" (as they are called) seldom last for more than a few days. In acute iritis, flakes of what looks like lymph may sometimes be seen clinging to the iris, while rarely the exudation

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may take the form of a reticulated grayish membrane, which has been likened to a spider's web. These changes, as a rule, are limited to a section of the lower portion of the iris, the other parts of that structure remaining free. At a later stage the exudations may organize into tough connective tissue, in which case one or several vessels are often observed running into the sheet from neighboring parts of the iris.

Exudation from the posterior surface of the iris must be assumed to have taken place whenever we find adhesions, or synechiæ, between the latter structure and the capsule of the crystalline lens. They are brought about by plastic material gluing, so to speak, the layer of pigmented epithelium to the lens-capsule at one or several points. It is important to bear in mind that the stroma of the iris takes no direct part in their formation. Their seat of election lies at or about what has been previously described as the uveal ring,—that is to say, the place where iris and capsule come naturally into the closest contact. They may be single or multiple, long or short, wide or narrow. Frequently one finds that, while the peripheral part of a synechia is pigmented, its attachment to the capsule is grayish white. The condition is spoken of as *exclusion* when the entire margin of the pupil is tied down by adhesions, so that all communication is cut off between the anterior and the posterior chamber. *Total posterior synechia* is said to be present when the hindermost surface of the iris, as a whole, is adherent to the lens-capsule, a state of things more commonly met with in cyclitis than in iritis pure and simple.[1]

It often happens that posterior synechiæ of recent formation are ruptured either by the spontaneous and incessant movements of the iris or by the correct and repeated application of a mydriatic. In either event, particles of uveal pigment and lymph are likely to remain attached to the lens-capsule, where they constitute permanent evidence of a past iritis. They take the form either of brown dots or of grayish points capped with pigment, and vary a good deal both in size and in number. If numerous, they commonly assume a more or less circular pattern. It is to be noted, however, that the diameter of the circle thereby described seldom exceeds three millimetres,—*i.e.*, it has less than the average diameter of the healthy pupil (from four to four and a half millimetres). This finds a natural explanation in the fact that synechiæ are formed during the height of the inflammatory process, at a time when the pupil possesses less than its normal width, or, in plain language, is contracted.

(*c*) Exudation into the pupillary area is most frequently shown by the

[1] The clinical methods of detecting synechiæ have been fully described in the introductory section, so that they need be only mentioned in this place. They are as follows: (1) simple inspection, aided (if need be) by focal illumination and the use of a magnifying glass; (2) observation of the movements of the pupil while the eye is alternately shaded and exposed to light; (3) the mirror test; and (4) the mydriatic test,—*i.e.*, ascertaining whether a one per cent. solution of sulphate of atropine causes the pupil to dilate regularly or otherwise.

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presence of lymph upon the anterior lens-capsule. A common and striking appearance is that of a narrow grayish-white fringe attached to some part of the free edge of the iris; it may, indeed, form a complete ring, although this is distinctly rare. At other times the pupil is occupied by a sheet of grayish material, which may or may not be pierced by one or more holes. This layer may have filamentous and reticulated attachments to the free edge of the iris, in which case the pupil will be irregular after the use of a mydriatic. On the other hand, it may be everywhere continuous with the iris, when *occlusion* is said to exist, a condition that must be carefully differentiated from that already described under the name of *exclusion*. The two may coexist, or they may be met with apart from each other. In the latter case the distinction between them becomes important, more especially from a prognostic stand-point. As pointed out by Fuchs, occlusion reduces sight in proportion to the density of the obstruction, but it does not entail any further mischief. Exclusion, however, invariably gives rise, sooner or later, to heightened tension. At the same time it is necessary to remember that the advent of glaucoma may be long delayed in such cases, because the functions of the ciliary body have been interfered with by the processes of inflammation, so that the aqueous humor is secreted in less than its normal amount.

(*d*) That more or less exudation into the aqueous humor accompanies every case of acute iritis is highly probable, although its character and amount vary much. For example, when the exudation contains relatively few cellular elements, the humor may be rendered merely cloudy. In consequence of this the anterior surface of the iris will seem duller than usual, while the pupil will have an ill-defined, hazy look. Again, particles of lymph of sufficient size to be recognized by the unaided eye may be diffused throughout the liquid, under which circumstances the deeper parts will, of course, be hidden from view. If by gravitation these particles sink to the bottom of the anterior chamber, then *hypopyon* will result, or an ill-defined mass may float, like a cloud, in the aqueous. It now and then comes about that vessels in the iris rupture during the height of inflammation, so that blood is effused into the aqueous humor. In slight cases the latter may be merely tinged, but sometimes it has all the appearance of liquid blood. This state of things is termed *hyphœma*. Lastly, an inflammatory effusion may coagulate, and thus give rise to what has been called a "spongy exudation" lying in the anterior chamber. This condition is exceedingly rare. Its appearance has been aptly compared with that presented by a dislocated lens (Schmidt-Rimpler).

5. **Disturbance of Sight, etc.**—When placed opposite the test-type, a patient with iritis will not infrequently declare himself unable to see the smaller lines, but after a little pressure from the surgeon he will read 6/6. This means, of course, that the eye is morbidly sensitive, so that the patient is unwilling at first to make the effort necessary for seeing accurately. But sight may be actually lowered by spasm of the sphincter

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iridis, by lacrymation, and by photophobia, conditions all of which may be present in the earlier stages of inflammation. Apart from these, however, vision seldom suffers much in iritis, always supposing that there are no mechanical reasons to the contrary, such, for example, as would be furnished by deposits upon the posterior surface of the cornea, by turbidity of the aqueous humor, or by lymph in the pupillary area. In all these cases the reduction in visual acuity will correspond (roughly, at all events) with the degree of obstruction that is present, a point of great importance in distinguishing iritis from irido-cyclitis.

A transient alteration in the refraction of the eye, as already noted, is occasionally present in acute iritis. Hypermetropia may thus diminish in amount, while emmetropia may pass over into myopia, and myopia may become augmented. Of course this can be scarcely recognized unless the refraction was known before inflammation set in, or unless one eye be alone affected, although the latter obviously assumes that the refraction of the two eyes was originally similar both in kind and in degree. Of the several explanations that have been advanced to account for this change, the most plausible is that which traces it to a spasmodic action of the ciliary muscle.

SYMPTOMATOLOGY OF CYCLITIS.

It has been already remarked that simple cyclitis is by no means a common disease. When occurring alone, it assumes, in the majority of cases, a subacute or chronic form of relapsing inflammation, its leading sign then being few or many dotted deposits upon the back of the cornea, a condition often, though incorrectly, spoken of as "serous iritis." As a rule, its symptoms are found combined with those of iritis, thus indicating that the surgeon has to deal with the mixed affection irido-cyclitis.

The existence of cyclitis as an independent malady, however, would be shown by the following signs and symptoms: 1. Pain, photophobia, lacrymation, etc. 2. Ciliary redness. 3. Stasis in the vessels of the iris. 4. Exudation of inflammatory products. 5. Disturbance of sight. 6. Tenderness of the ciliary region. 7. Variations in the tension of the eyeball. 8. Miscellaneous signs and symptoms, as, for example, blanching of the eyelashes and eyebrows, and entoptic phenomena.

In pursuance of the plan adopted when dealing with the symptoms of iritis, the foregoing may be next taken up and discussed one by one.

1. **Pain, Photophobia, Lacrymation, etc.**—The pain of cyclitis usually stands in direct proportion to the severity and especially to the acuteness of the inflammation. Very similar considerations apply to lacrymation and photophobia. A certain amount of systemic disturbance will at times be present, but only in very acute cases. This will be indicated by a rise in temperature, mental irritability, sleeplessness, a flushed face, a quickened pulse, a furred tongue, nausea, or vomiting.

2. **Ciliary Redness.**—The injection present in cyclitis has the same general characters as that already described in iritis; that is to say, it

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mainly involves the episcleral twigs of the anterior ciliary arteries, which form a zone from two to five millimetres in width around the cornea. At the same time it should be noted that there are two respects in which it offers some points of contrast to the latter. In the first place, the congestion of cyclitis tends to be violet rather than red in color; secondly, it is often unequal at different parts of the circle, or, in other words, shows a tendency to what has been called "patchiness." Cyclitic redness may be trivial or marked. For example, in slight cases of "serous iritis" there may be no redness whatever, or, at most, a faint ciliary blush which comes on after examination with the ophthalmoscope. In cyclitis from injury, on the contrary, it may exist in a marked degree, although under such circumstances it is often enough hidden by the chemosis that may be present. The upper lid, as a rule, will in this case be swollen, a change that generally begins at its free edge and later creeps over the rest of its extent. Lastly, in severe cyclitis the whole vascular system of the front of the eye is almost sure to be engaged, so that no diagnosis can then be safely made from the kind of injection that is present.

3. **Stasis in the Vessels of the Iris.**—It sometimes happens that, owing to inflammatory changes in the ciliary body, the outflow from the iris is impeded, in which case the veins of that membrane may become tortuous and visible to the unaided eye, a change generally more marked towards the periphery than elsewhere. This venous stasis may to some extent modify the color of the iris, rendering it reddish or reddish gray. Apart from this and also from actual iritis, a remarkable uniform grass-green hue is occasionally observed in cases of simple cyclitis, which may possibly be due to blood from the inflamed parts gaining access to the aqueous humor and in that way modifying the apparent color of the iris.

4. **Exudation of Inflammatory Products.**—The existence of inflammatory deposits in various parts of the eye constitutes by far the most trustworthy sign of cyclitis. Besides occurring into the tissues of the ciliary body, they are also cast off from its inner surface, thus gaining admission to the anterior, posterior, and vitreous chambers, where they may be recognized by the surgeon. They may be either plastic or purulent in nature. For convenience of description, these inflammatory exudations may be discussed under two heads: first, those met with in front of the zonule of Zinn; secondly, those lying behind that structure.[1]

Inflammatory exudations which leave the ciliary body in front of the zonule of Zinn pass into the posterior chamber, where they mingle with the aqueous humor and are conveyed by that fluid into the anterior chamber. The humor in this way becomes more or less cloudy, and its contained particles may be deposited at the lower part of the space, where they constitute the appearance clinically known as hypopyon. The hypo-

[1] It must be borne in mind, however, that the meshes of the zonula itself may be infiltrated by leucocytes or epithelial cells, derived from the inflamed ciliary body.

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pyon of cyclitis is peculiar in that it appears and disappears at short intervals of time. Again, if the exudation be rich in fibrin, and rapidly poured out, the so-called spongy deposit may be met with. Hyphæma, also, is seen when vesssels in the inflamed ciliary body rupture.

In addition to this, the suspended particles may be deposited upon the posterior surface of the cornea, upon the iris, or upon the exposed portion of the anterior capsule of the lens.

In the first-named position they form the appearance familiar to surgeons as *keratitis punctata*, *hydromeningitis*, *aquo-capsulitis*, *descemetitis*, all of which titles, one may remark in passing, are based upon a mistaken pathology. *Serous iritis* also is another expression that has been employed, but that term lies open to more than one objection. In the first place, exudations are found when the iris, so far as we can tell, is perfectly free from inflammation; secondly, it is clearly a misuse of terms to speak of formed deposits as "serous." It would be well, therefore, to abandon the expression altogether, and to replace it by one more in accord with the known facts of the case, such as "simple cyclitis."

Recognizable deposits are sometimes ushered in by a faint localized dulness of Descemet's membrane, a change which upon close examination is found to be made up of "cross-hatched" lines or "fingers" of grayish opacity spreading from the circumference towards the centre of the cornea. These gray lines, also common after traumatism (accidental or operative), have been accounted for in various ways,—as, for example, by a widening of the tissue-spaces in the deeper layers of the cornea (Becker), by folding of Descemet's membrane (Hess), or by an œdema of the posterior layers of the cornea (Spicer). They may exist by themselves, but more commonly are associated with, or followed by, punctate dots upon the posterior elastic lamina of the cornea.

The dots themselves may be few or many in number. When of small size, it is necessary to use a magnifying glass and artificial light in order to identify them; when large, they may, on the contrary, be visible to the naked eye. They present the peculiarity of being grouped (doubtless in obedience to the laws of gravitation) mainly upon the lower half of the cornea, while for a similar reason the larger ones usually lie below the smaller. In this way it comes to pass that they commonly assume the form of a triangle, the base of which is directed centrifugally. Arlt demonstrated experimentally that these punctate deposits were actual precipitations from the aqueous humor. He found that the base of the triangular figure was always directed towards the side to which the patient's head was turned. Thus, if the patient lay upon his right side the spots would become so grouped that, while the base of the triangle in the right eye corresponded with the temporal side of the corneal margin, that in the left eye corresponded with its nasal side.

The individual spots have a sharply cut, grayish-white look. They have been likened, with more aptitude than elegance, to drops of cold

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gravy fat. As James Wardrop,[1] the first to describe them, remarked, they "do not resemble any of the common forms of speck, but have a mottled appearance; and around the more opaque white central points of these specks there is a kind of disk, very like what is to be perceived in some agates, and what are commonly called the eyes of pebbles." Wardrop[2] also pointed out that many of the deposits flow away with the aqueous after paracentesis has been performed, and spoke of "an instantaneous restoration of the transparency of the anterior chamber" as following the evacuation of that humor.

The deposits, when numerous, cause very serious interference with sight, which, as insisted upon by William Mackenzie,[3] may show remarkable and rapid fluctuations. That author related particulars of an instructive case in which the spots "partially appeared and disappeared, so that the patient was worse in the morning, when most of the spots were observed, and better towards the evening, when those at the upper part of the cornea had considerably diminished." The obvious explanation of this striking fact is that the erect posture maintained by the patient during the daytime caused the exudations, formerly diffused over the whole internal surface of the cornea, to fall below the pupillary area of that membrane.

Similar but larger grayish-white spots may be occasionally recognized upon the anterior surface of the iris or upon the pupillary area of the lens-capsule. They are, as a rule, by no means numerous. Should these deposits encroach upon the inner edge of the iris they may give rise to an unusual kind of posterior synechia, which takes the form of a grayish band connecting the anterior surface of the latter with a neighboring part of the capsule. Should they be situated towards the sinus of the anterior chamber, they may, on the other hand, constitute a bond of union between the ciliary zone of the iris and the posterior surface of the cornea; that is to say, peripheral anterior synechiæ may result. The condition, probably, is always a sign of cyclitis, antecedent or existent.

It sometimes happens that a layer of inflammatory exudation becomes precipitated, as it were, into the pupillary area, thus giving rise to "occlusion of the pupil," a condition already fully described when speaking of iritis.

Another result of cyclitis—namely, total posterior synechia—has been touched upon more than once in the foregoing pages. This very serious sequel is brought about in the following way. Plastic material, furnished by the inflamed ciliary body, comes to lie between the uveal surface of the iris, in front, and the anterior capsule of the crystalline lens and the zonula, behind; so that the posterior chamber is more or less occupied by exudation. This is followed by organization, in consequence of which the fluid exudation is ultimately replaced by fibrous material, which inseparably

[1] Essays on the Morbid Anatomy of the Human Eye, 1818, vol. ii. p. 8.
[2] Medico-Chirurgical Transactions, 1813, vol. iv. p. 162.
[3] A Practical Treatise on the Diseases of the Eye, 4th ed.

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unites the above-named structures. The iris, as it were, gets moulded to the front of the crystalline lens, so that the anterior chamber becomes deeper than normal. This change is particularly noticeable at the periphery, more especially when pathological adhesions have occurred between the root of the iris and the ciliary processes. Total posterior synechia is recognized clinically in three ways: (1) by the fact that the iris, even when exposed to the strongest light, possesses no action whatever; (2) by the existence of degenerative changes in the iris, shown by local or general thinning, by irregular pigmentation, or by small rents in its substance; (3) by alterations in the appearance of the anterior chamber, such as increased depth, especially at its circumference (*retraction of the iris*).

Deposits leaving the ciliary body posterior to the zonule of Zinn pass, as a rule, into the fore part of the vitreous humor, which they render merely hazy or actually cloudy, according to their size and number. If the inflammatory process remain unchecked, they may permeate the entire humor. They not infrequently form a specially dense layer immediately behind the crystalline lens. These deposits, when marked, may be recognized by the ophthalmoscope as forming floating or fixed opacities in the vitreous humor; indeed, when of large size and lying far forward, they may be seen by focal illumination. They give rise, of course, to serious imperfections of sight. In their further history the deposits either become absorbed, wholly or in part, or else organize into connective tissue. In the latter case the bands and membranes thereby produced may in course of time give rise by their traction to disastrous results, such as opacity or dislocation of the lens, separation of the ciliary body from its matrix, detachment of retina or choroid. These changes are generally followed by *phthisis bulbi*, a melancholy condition in which there is loss of sight and lowered tension, the eye being smaller than natural and distorted in outline. Atrophic globes are often painful or tender, besides which they constitute a fertile cause of sympathetic ophthalmitis, more especially if they contain plates of cartilage or bone in or upon the choroid or upon the anterior surface of the ciliary body.

5. **Disturbance of Sight.**—It may be laid down almost as a general rule that every case of cyclitis is accompanied by some disturbance of sight. The reasons for this statement must be abundantly evident from what has gone before. There will, therefore, be no necessity to offer any further remarks upon the matter in this place.

6. **Tenderness of the Ciliary Region.**—Tenderness of the ciliary zone of the sclera is not ordinarily present in simple iritis, so that its existence in marked form may be regarded as diagnostic of cyclitis, or at least of cyclitic complication. At the same time the fact must be insisted upon that some forms of cyclitis are not accompanied by tenderness of the eyeball. The surgeon, however, should always make a point of ascertaining its presence or absence by the methods previously explained (see page 265). Although tenderness may be present at all points of the zone, yet it may, on the other hand, be limited to one particular region, as, for example, the

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superior nasal or superior temporal quadrant of the eyeball. It is now and then so marked that the lightest touch upon the ciliary region causes the patient to start back instinctively, exclaiming that he "cannot bear the pain."

7. **Variations in the Tension of the Eyeball.**—No doubt some alteration in tension invariably accompanies cyclitis, although in slight cases the variation from the normal may be so small that the finger is unable to appreciate it. In severe cases, however, there is no room for doubt. Broadly speaking, in the early stages of the inflammation tension is heightened and in the final stages lowered, while in the intermediate stages it fluctuates according to circumstances, being now raised, now lowered. Alterations of the kind described possess very great importance from a diagnostic point of view. Thus, increased pressure occurring in a young person and associated with external signs of inflammation is far more likely to be the result of cyclitis than of any other malady. This fact should be carefully borne in mind, since there are good reasons for believing that, by inexperienced observers, cyclitis is sometimes mistaken for glaucoma.

8. **Miscellaneous Signs and Symptoms.**—Whitening of the eyelashes and eyebrows has been observed to follow cyclitis, especially when due to sympathetic mischief. The change may pick out, so to speak, certain tufts of hair, or it may, less commonly perhaps, affect the brows and lashes as a whole. It has been suggested that the blanching may be accounted for on the grounds of mere coincidence; that no real connection exists between the two conditions. Sufficient cases, however, have now been recorded to render that explanation improbable.

One additional symptom remains to be mentioned, namely, the entoptic phenomena sometimes complained of by patients. These assume subjectively the most curious and fantastic shapes; they may be likened, for example, to flies, cobwebs, lace curtains, beetles, octopi, "floating blacks," "dancing squares," rapidly rotating rings of colored fire, and the like. They are capable of compromising vision seriously, and compel the sufferer to twist his head about in various ways in the endeavor to avoid the obstruction they give rise to. In general terms it may be said that they are suggestive of deep-seated lesions in the vitreous or the retina. When persistent, therefore, they should be regarded as of evil omen.

PROGRESS, DURATION, AND TERMINATION.

As regards its course, an iritis or cyclitis may be acute, subacute, or chronic. Acute iritis speedily reaches its acme; the ciliary redness then usually fades, pain becomes less, and the pupil (unless tagged down by adhesions to the lens-capsule) reacts promptly and satisfactorily to atropine. Within from four to eight weeks all signs of inflammation have usually disappeared; indeed, there are exceptional cases where a few days suffice for the cure of the affection. Recovery may occur without residual adhesions. More commonly, however, an attentive examination will disclose the existence of one or more synechiæ, or of spots of pigmented lymph

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upon the anterior capsule of the lens (see page 271). Sometimes, too, the color of the iris may show permanent changes (see page 258). As will be explained in detail later, certain instances of acute iritis manifest a marked tendency to recur under the exciting influence of trifling causes, such as exposure to cold or wet. Subacute cases, as implied by the name, show slighter signs of inflammation than the acute, but run, as a rule, a more tedious course; moreover, they practically always entail synechiæ or other permanent sequelæ. Pure cyclitis—a rare affection—is essentially chronic. It is very prone to relapse, and is marked simply by a faint ciliary blush, a vitreous haze, and keratitis punctata. In mild cases both the vitreous humour and the cornea may clear, although as regards the latter a few pigmented spots often remain for years to tell the tale of former mischief. Now and again iritis is chronic, either from the outset or after passing through a preliminary acute or subacute phase. It is then characterized by some irritability of the eye, together with a sluggish and slightly contracted pupil; although the iris may or may not be appreciably thickened, yet exudative changes are generally present.

Inflammation of the iris or ciliary body may be associated with copious hemorrhage into the anterior chamber. By some authors this is regarded as constituting a definite morbid entity, but others look upon the bleeding as a complication merely. To our mind, there is nothing specific about this so-called *hemorrhagic iritis;* in point of fact, extravasation of blood may occur in any acute plastic inflammation affecting either the iris or the ciliary body.

Under the name of *quiet* or *insidious* iritis, a series of cases has been described in which the classical signs of inflammation are almost or altogether wanting, failure of sight being the first symptom noticed by the patient. Under these circumstances the surgeon may find many adhesions, or even an "excluded" or "occluded" pupil. From an analysis of thirty-seven cases, Jonathan Hutchinson, Jr.,[1] concludes that while this form of disease "is most commonly due to congenital syphilis, sympathetic inflammation, or inherited arthritic (rheumatic or gouty) tendency, it is very exceptional in connection with acquired syphilis or the ordinary rheumatic form of iritis." This statement, however, which appears to cover most of the known causes of iritis, fails to advance materially our knowledge of the subject. Our own experience is that "quiet" iritis is generally seen in patients who exhibit no marked constitutional tendency other than extreme debility.

As already stated, an acute iritis may recover so perfectly that the most careful scrutiny of the eye will fail to discover a single sign of former inflammation. This, however, is comparatively rare; as a rule, certain traces remain which may be readily recognized even after the lapse of

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[1] Transactions of the Ophthalmological Society of the United Kingdom, vol. viii. p. 117 *et seq.*

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many years. These are two in number,—namely, *posterior synechiæ*, and *pigmented spots of organized lymph upon the anterior capsule of the lens.* The former, one must recollect, sometimes undergo spontaneous rupture in consequence of the movements of the iris, but they must be pretty recent in order that this may take place. The grayish spots, as noted previously, represent the capsular attachment of broken adhesions, and often present an approximately circular arrangement. They appear to be permanent. Subacute and chronic iritis, as well as irido-cyclitis, generally entail lasting results, so that the functions of the eye become impaired or altogether lost. Although these have been already touched upon, they may, nevertheless, be grouped together in the following way:

1. Few or many ordinary posterior synechiæ.
2. Total posterior synechiæ.
3. Adhesions of the iris to the cornea (rare).
4. "Occlusion" of the pupil.
5. "Exclusion" of the pupil and secondary glaucoma.
6. Irregular pigmentation and atrophy of iris.
7. Lenticular opacities.
8. Secondary corneal changes, detachment of retina, ossification of choroid, and shrinking of the eyeball.

PATHOLOGICAL ANATOMY OF IRITIS AND OF CYCLITIS.

From a pathological point of view there is no essential difference between mere hyperæmia and commencing inflammation of the iris, since both conditions are characterized by dilatation of the blood-vessels. In actual iritis, however, the process does not end there; diapedesis occurs, the meshes of the iris become distended by effused fluid, and at a later stage the cellular elements of the parenchyma proliferate. Small hemorrhages often occur into the iris, the pigmented cells of which show various alterations. As the net result of these changes, the iris gets thickened, while its tissues become pervaded by few or many small, round cells containing one or more nuclei. This exudation may remain confined to the iris, or it may, on the other hand, pass into surrounding parts, as the anterior chamber or the pupil. In its further course, it may disappear completely or become converted into connective tissue, as will be explained in detail later in these pages. Cyclitis is associated with similar changes, and the products of inflammation undergo similar transformations. It may be noted that the ciliary muscle is seldom, if ever, the part first affected by inflammation, probably because its vascular supply is relatively less abundant than that of the ciliary body. At a later stage, however, its muscular fibres may be found to have undergone fatty or other degenerative changes.

Plastic iritis, like any other inflammation, is characterized primarily by hyperæmia of the blood-vessels and then by exudation of cellular and fibrinous products. The vessels may be so engorged with blood as to rupture and discharge their contents into the anterior chamber (*hyphœma*).

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The exudation consists of fibrin, entangling few or many leucocytes, which contain one or more nuclei. It may be found in, upon, or behind the iris, or upon the pupillary area of the lens-capsule. This deposit, as stated above, may undergo resorption or organization. In the latter event, the fibrin forms a sort of matrix in which the connective tissues are laid down. Some of the mononuclear cells become elongated and spindle-shaped, and these "fibroblasts," as they are termed, remain connected with one another either laterally or by pointed processes; blood is supplied by offshoots from existing vessels. The polynuclear cells disappear, wholly or in part. The fibrin is eventually replaced by connective tissue, which, although at first rich in cells and vessels, later becomes denser and less vascular. In short, fibrous or cicatricial tissue has now been produced.

The effects produced by this newly formed tissue depend upon its position. Thus, if the substance of the iris be invaded, fatty and hyaline degeneration and atrophy will ensue to an extent that will vary both with the amount and with the position of the exudation. For example, in slight cases one or more spots may alone suffer, whereas in severe ones everything, save the sphincter muscle and the uvea, may be replaced by irregularly pigmented connective tissue. Calcareous masses may be occasionally seen, as in a case described by Panas, where a bony nucleus lay between the uveal layer and the "fibres of Henle." If the deposit occurs at the edge of the pupil or upon the hinder surface of the iris, then posterior synechiæ will be met with. These may be single, multiple, or total; and, as already stated, when the entire uveal zone is adherent to the capsule, the pupil is said to be "excluded." In all these conditions the uveal layer of the iris is cemented to the capsule of the lens by means of newly formed material which contains varying proportions of round and spindle cells, together with vessels. In accordance with the rule, this tissue becomes at a later period more dense and less vascular. "Exclusion" of the pupil, as pointed out, sooner or later entails as its necessary consequence a bulging (or "crater-shaped") iris, the result of pressure by fluid pent up behind the iris by a mechanical obliteration of the so-called filtration-angle of the anterior chamber. The iris occasionally becomes so convex as to touch the posterior surface of the cornea, and in that way one kind of anterior synechia may be brought about. It is obvious that this protrusion cannot occur when the posterior surface of the iris is tied down universally to the lens-capsule, as in the condition spoken of as *total posterior synechia*. Sometimes, however, the structures named are adherent at certain places merely, under which circumstances localized bulgings of the iris may be seen (*gibbous iris*).

If not remedied by operation, *exclusion* becomes associated with atrophic changes in the iris, which in that event loses its reticulated appearance, becomes thin, and has a "washed-out" look. Microscopically, it is found to be represented by a layer of compact tissue which contains scarcely any trace of the original structure. Its vessels may be the subject of hyaline

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degeneration or may be obliterated. When the pupil is blocked by exudation, *occlusion* is said to exist. This condition is sometimes combined with that just described,—namely, *exclusion*,—although the converse is perhaps more frequently the case. At an early stage the exudation is found to be made up of fibrin and round cells, which latter organize into a definite connective-tissue membrane. This sheet may be incomplete,—that is, perforated by few or many small holes,—sieve-like, so to speak. It may be connected with the border of the iris by separate processes, but it is more often found to overlap, as it were, the edge of that structure. Vessels pass into the obstruction from the iris, and may be large enough to be recognized as such by the naked eye. The pupil may suffer various distortions by the contraction of the newly formed tissue. We must distinguish this form of occlusion from another in which the pupil (probably during sleep) gets blocked by fibrinous material deposited from the turbid aqueous humor. The membrane formed under these circumstances adheres but loosely to the edge of the iris, and is hence readily detachable by surgical means. Lastly, it should be noted that small rents in the iris (or, more rarely, actual irido-dialysis) have been observed in certain instances where the pupil became "occluded" in early life. The explanation, of course, is that during the growth of the eye the tied-down iris has become stretched and eventually torn.

According to Adolf Alt,[1] who has had an opportunity of examining such a case with the microscope, the so-called "spongy exudation" is brought about in the following way. As a first step, numerous hemorrhages occur into the parenchyma of the iris. The cellular elements of the effused blood then undergo fatty degeneration *in situ*, but the fluid parts gain access to the anterior chamber, where the fibrin coagulates and gives rise to the peculiar and characteristic appearance already likened to an opaque and dislocated crystalline lens. The resemblance, it may be added, is rendered more realistic by the fact that absorption of the gelatinous mass commences in the parts nearest the cornea, and proceeds in a sort of concentric fashion.

Purulent iritis includes a number of widely different conditions. For example, the hypopyon so commonly associated with rheumatic or specific irido-cyclitis has, from a pathological point of view, as much claim to be called "purulent" as the wide-spread and fatal form of inflammation that follows the entrance into the eye of septic material. Broadly, however, a purulent exudation is distinguished from a plastic one by the fact that it contains many cells floating in a fluid medium. In marked cases the pathological changes occur with considerable rapidity. Thus, diapedesis and effusion of red blood-corpuscles are quickly followed by division of the fixed cells of the iris. Some of the emigrated corpuscles gain access to the aqueous humor, where they constitute *hypopyon*. At the same time

[1] Lectures on the Human Eye, p. 86.

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the iris loses all proper distinction of structure, becomes greatly thickened, and is converted into what, under the microscope, appears to be a mere mass of leucocytes.

It has been already pointed out that cyclitis may entail results similar to those of iritis, so that there will be no need to repeat what has been already said upon that subject. There are, nevertheless, two points that deserve separate mention: first, the deposits of so-called keratitis punctata; secondly, the natural history of what are termed, aptly enough, "cyclitic membranes."

Keratitis Punctata.—It was formerly thought that the grayish spots of keratitis punctata resulted from proliferation of the endothelial cells that line Descemet's membrane (Schweigger, Ivanoff, etc.). This view, however, is almost entirely given up at the present time. The little masses are now regarded as deposits which are thrown, as it were, against the posterior surface of the cornea from the aqueous humor, and which come in the first instance from an inflamed ciliary body. Strictly speaking, they are signs, therefore, not of iritis, but of cyclitis. Histologically, they consist of collections of round, nucleated cells, cemented together by a varying amount of fibrinous material, containing small granular particles. Tiny deposits of pigment, mainly derived from the outer layer of the pars ciliaris retinæ, are constantly present, enclosed within the round cells or lying free in the fibrin. The endothelium appears to take no active part in the formation of these deposits, although its cells may be displaced by the masses and may then undergo degenerative changes.[1] Descemet's membrane remains intact. Herman Snellen[2] has recently declared that the dots contain micro-organisms, capable of being grown upon agar-agar. But his observations, which were carried out upon a limited number of cases only, will be mentioned later. (See page 302.) The punctate deposits usually disappear step by step with their cause, but small deposits of pigment lying upon the posterior surface of the cornea may sometimes be recognized months after inflammation has come to an end. Moreover, the deposits may become organized into connective tissue. This is well exemplified in the irido-cyclitis of hereditary syphilis, where they may be now and then replaced by a bluish-white triangular plaque. The punctate deposits occasionally found on the anterior capsule of the lens, or upon the front of the iris, have a similar histological structure. Lastly, it should be added that groups of leucocytes are not infrequently found in or about the angle of the anterior chamber in cases of serous cyclitis.

Cyclitic Membranes.—Cyclitic membranes appear to be formed in the following way. The inflamed ciliary body casts off a fibrinous exudation, which, together with pigmented cells from the pars ciliaris retinæ, may find

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[1] See Lawford, Royal London Ophthalmic Hospital Reports, vol. xii. p. 298; Ridley, ibidem, vol. xiv. p. 237.

[2] Ophthalmic Review, vol. xiii. p. 259.

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its way into the vitreous, or into the posterior or anterior chamber of the eyeball. This exudation may then become absorbed, or it may, on the contrary, undergo organization. In the latter event, some of its contained leucocytes are converted into fibroblasts, while others are destroyed. The formative cells increase in number, their nuclei enlarge, and the intercellular substance becomes fibrillated. Meanwhile blood-vessels of new formation penetrate the tissue from neighboring parts. As the outcome of the changes thus briefly outlined, granulation tissue is ultimately replaced by connective tissue. According to Alt, however, the retinal layer of the ciliary body takes an active share in the formation of these membranes, or may even be their starting-point. That author states that the cells proliferate, and finally become converted into connective-tissue fibres. The pigmented portion of the pars ciliaris retinæ, too, he says, may suffer similar changes, so that a series of cylindrical and branched processes may be seen growing into the cyclitic membrane. He compares the section of these excrescences with "the glandulæ tubulosæ or the epithelial cylinders of an epithelioma." The stress of these various alterations, be it noted, falls mainly upon the smooth part of the ciliary body. Treacher Collins believes that the cylindrical overgrowths result from a hyperplasia of the tubular glands described by him as existing in the pigmented, or outer, portion of the pars ciliaris.

Tubular outgrowths certainly often extend into the vitreous from the pars ciliaris retinæ. But it is difficult to see how they can be direct products of the proliferation of the external (pigmented) stratum alone. It seems reasonable to attribute them to overgrowths from the columns or rod-like cells of the internal stratum, into which pigment granules have been carried from the altered and degenerated external layer. According to this view, therefore, the pigmentation is a secondary process merely.

The formed cyclitic membrane, then, is made up of fibrillated connective tissue, which contains cells of various kinds, cylinders more or less pigmented, blood-vessels, fibrin, and amorphous material. The vessels, which are extensions from those of surrounding parts, frequently rupture, so that irregular lumps of pigment may be found scattered here and there throughout the newly formed tissue. At a later stage cyclitic membranes may suffer any or all of the metaplastic changes to which the connective-tissue series is liable. This is equal to saying that fatty degeneration may take place, or that calcareous or osseous deposits may be observed. Apart from this, the membrane, like all recent connective tissue, may undergo progressive contraction, and the nutrition of the neighboring parts may thereby be affected in the most disastrous way. Thus, small cysts may be met with lying between the posterior part of the pars ciliaris retinæ and the subjacent pigment layer, or the retina may become detached, or the choroid separated from the sclera. It is by no means uncommon for a layer of newly formed material to pass across the vitreous chamber, swathing, so to speak, the posterior

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surface of the crystalline lens. By subsequent shrinkage this may thrust the lens and central part of the iris towards the cornea and at the same time drag the root of the iris backward and the ciliary body from its matrix. But it would serve no useful purpose to describe all the possible modifications that may ensue. It must suffice to remind readers that these have been enumerated in an earlier part of this article, to which they are referred for further details (see page 280).

ETIOLOGY.

It has been computed by various writers of authority that iritis furnishes from two and three-tenths per cent. to four per cent. of all ophthalmic cases. The disease is certainly as uncommon during the first fifteen years of life as it is during old age, and is most frequently met with between the twentieth and the fortieth year. There appear to be good grounds for the current belief that it affects men in greater relative proportion than women.

Syphilis is, without doubt, the most common predisposing cause of iritis. Most authors credit the venereal disorder with fifty per cent. of all cases, but that estimate is too low, especially if hereditary syphilis be taken into account, and the proportion probably lies near sixty per cent. Rheumatism is responsible for most of the other cases, so that we shall not be far wrong if we lay thirty per cent. to the score of that ailment. The remaining ten per cent. are due to various causes, of which injuries, gonorrhœa, gout, diabetes, and malarial and other fevers are probably the most important. We may vary this statement by saying that of every ten cases of iritis, six will in all likelihood be the result of syphilis, inherited or acquired, while three will be caused by rheumatism, and one by some of the other conditions enumerated above.

The presumptive etiology of iritis will depend to some extent upon the age of the patient. Thus, in infancy the ailment is generally due either to inherited syphilis or to the results of ophthalmia neonatorum; in older children it is caused by inherited syphilis or by traumatism; in adults, acquired syphilis, rheumatism, or gonorrhœa will, as a rule, be found to lie at the root of the matter; whilst in old people it is usually observed as a sequel to some operative interference with the eyeball, such as cataract extraction.

Although in a majority of cases, then, some constitutional condition predisposes to iritis, yet it must be remembered that in many instances, if not in all, an exciting cause is also present. For instance, many rheumatic patients definitely date their first attack of iritis from exposure to cold, from prolonged reading, or from a slight injury. A similar statement is certainly true of syphilis. A surgeon must be careful, therefore, not to mistake one for the other, a warning that is all the more necessary since the sufferer himself may be fully alive to the exciting cause, but may conceal or may be unaware of the predisposing cause of his iritis.

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It was formerly taught that the mere inspection of an inflamed iris would enable the surgeon to arrive at a definite conclusion with regard to etiology, so that a good deal of stress was naturally laid upon the clinical appearance of the eye. This idea has now been largely modified. While there are instances in which the nature of an iritis may be diagnosed in that way, there are at least an equal number of cases in which such a test gives no indication whatever as to the constitutional cause of the inflammation. As a matter of fact, a decision upon the latter point must be based in iritis, as in other affections, upon many considerations, of which the most important are a knowledge of the previous history and condition of the patient, his present state, the salient features of the ocular attack, and the effects of treatment. In other words, the anatomical diagnosis has come to occupy a more or less subordinate position, and is appealed to, as a rule, merely as confirming or negativing an opinion arrived at from other data.

We may next pass onward to discuss the various types of iritis and cyclitis, indicating as we proceed the main clinical features of each. In some of these forms the ciliary body appears to be mainly involved; in others the iris bears the brunt of inflammation; while in still a third class both structures are equally implicated. It would be almost impossible, however, to separate them from one another, and so a common description must suffice.

The older method was to classify an iritis or a cyclitis into *serous*, *plastic*, or *purulent*, according to the supposed nature of its inflammatory products. While this nomenclature is not devoid of practical convenience, there is, nevertheless, at least one serious objection to its employment, namely, that a given case may at one period in its life-history show a serous, and at another a plastic or even a purulent, exudation. In the course of the following pages, therefore, an etiological classification will be adopted.

CLASSIFICATION OF IRIDO-CYCLITIS.

Primary.—Syphilitic, rheumatic, gonorrhœal, gouty, diabetic, herpetic, malarial, climacteric, cerebral, scrofulous, tubercular, traumatic, post-febrile, cachectic, idiopathic.

Secondary.

Sympathetic.

Primary.—*Syphilis.*—Iritis, as is well known, occurs both in hereditary and in acquired syphilis. In the former it is seen under two conditions: (*a*) during the first eighteen months of life; (*b*) at a later period, when it is met with either alone or along with interstitial keratitis.

(*a*) Infantile iritis was recognized and described by William Mackenzie,[1] but more recently Mr. Jonathan Hutchinson[2] has given a careful account

[1] A Practical Treatise on the Diseases of the Eye, 4th edition, London, 1854, p. 546.

[2] Syphilis, London, 1889, p. 239.

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of it. According to the last-named observer, the malady commences at the average age of about five months, affects females more often than males, attacks one or both eyes, and, although generally associated with a pretty free exudation of lymph, is not marked by much redness of the eye. The cornea, as a rule, remains perfectly clear throughout the attack, and the ailment is attended by few of the more severe symptoms met with in adults. Most of the cases show one or more of the signs of hereditary taint, as cachexia, psoriasis-like or other cutaneous eruptions, aphthæ or sores about the mouth, condylomata around the anus, or "snuffles." The prognosis is favorable, provided mercurial treatment be adopted in good time. Mr. Hutchinson believes that this form of iritis "is amongst the rarest of the symptoms of hereditary syphilis."

(*b*) The iritis met with in older subjects, as already stated, may occur alone or along with interstitial keratitis. In the former case the inflammation, as a rule, appears to begin in the ciliary body and to spread to the iris at a later stage. Strictly speaking, therefore, it is an irido-cyclitis. Its recognition may be difficult, because in many instances no very obvious signs of hereditary syphilis can be made out. For example, it not infrequently occurs in well-grown and finely developed patients, whose teeth are above suspicion, and whose faces bear none of the appearances held to be characteristic of an inherited taint. At the same time, a proportion of the cases are deaf, while a certain number show other eye-symptoms, as, for example, disseminated choroiditis, which are generally attributed to syphilis. The family history is of great importance, and, when it can be obtained with any approach to trustworthiness, may often clear up the etiology of a doubtful case. Sometimes, too, a direct admission of syphilis may be elicited from the parents, who may themselves manifest specific ocular or bodily affections.

There are facts which indicate that this irido-cyclitis is closely allied to ordinary interstitial keratitis. Thus, a patient may manifest one condition in his right and the other in his left eye; or in one and the same eye the former may gradually merge into the latter.

Although varying within wide limits as to age, yet this form of specific irido-cyclitis appears, upon the average, at about twenty-one. Both eyes are generally affected, but the disease seldom attacks them simultaneously. The eye becomes irritable, unable to bear the light or to get through much work. Ciliary congestion, purplish in hue and patchy in distribution, is present. The iris reacts to light imperfectly or not at all, although posterior synechiæ are seldom observed in mild cases. Sometimes the pupil may get blocked by a layer of grayish material, which, since it adheres but loosely to the edge of the iris, may be assumed to have been thrown down from the aqueous humor. The cornea may show that remarkable appearance formerly spoken of by us as "cross-hatching" (see page 275), while punctate deposits of various sizes are constantly present. A common condition is that of a dense bluish-white plaque of triangular outline, which

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lies deep in or behind the lower half of the cornea, and which results from organization of the punctate deposits. Tension is essentially fluctuating, being now above and now below normal, a series of changes that may take place with great rapidity. Secondary glaucoma forms a common sequel. Lastly, disseminated choroidal changes are now and again observed.

What has been said regarding the late iritis of inherited syphilis may be summed up, then, as follows:

1. It occurs at the average age of twenty-one years. 2. It often affects those who possess a fine physique and show no other signs of hereditary taint. 3. In a majority of cases it involves both eyes. 4. It usually takes the form of a serous irido-cyclitis; in other words, it is characterized by dots or a white triangular plaque upon the posterior surface of the cornea, and is apt to lead to secondary glaucoma. Rapid variations in tension also are generally observed. 5. Less commonly it is of gummatous or of plastic type.

Although the existence of irido-cyclitis may be sometimes demonstrated during the course of an interstitial keratitis, yet more commonly its signs are not recognized until the cornea has commenced to clear. This fact renders it somewhat difficult to say whether the first-named affection should be regarded as coexistent with or secondary to the keratitis. It may be assumed, however, that cyclitic changes are present in every case of parenchymatous keratitis that shows rapid variation in tension, marked tenderness of the eyeball, or swelling of the free edge of the upper lid; in short, whenever the symptoms of inflammation reach an unusually high pitch.

In acquired syphilis, as in hereditary disease, irido-cyclitis is met with at two periods: (*a*) within the first nine months after infection; (*b*) at a much later period of the disease.

(*a*) The usual form of specific iritis is distinctly a secondary phenomenon, and, for obvious reasons, is almost exclusively confined to adults. It generally makes its appearance within the first nine months after infection, thus roughly coinciding in point of time with the sore throat, the symmetrical ulcers of the tonsils, and the earlier cutaneous eruptions. Its dependence upon syphilis, however, may or may not be easy to make out. Thus, it is a simple matter to assure oneself of the relationship, provided the patient shows other secondary symptoms, or gives a clear personal account of an infecting chancre. If the existence of a venereal complaint be denied, the surgeon must base his conclusion upon other evidence, such as the appearance of the inflamed eye or even the effects of mercurial treatment. It is not uncommon for patients to say that the trouble began after exposure to cold or to wet. In other words, they are fully aware of the immediate exciting cause of their ailment, while quite possibly they know nothing whatever of its predisposing cause. Hence full inquiry should be made into the personal history of every case of iritis, and,

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whenever possible, the patient should be thoroughly examined, so as to find out whether he has any signs of secondary syphilis.

The iritis of secondary syphilis is nearly always of plastic, or rather adhesive, type. It has comparatively little tendency to recur. At the outset, inflammation is usually limited to a single eye: Boeck, among one hundred and twenty-six patients, had but two in whom the disease commenced in both eyes simultaneously. According to Ammon, the left generally suffers before the right eye; but in a majority of the cases the ailment eventually becomes bilateral. Symptoms show a wide diversity as regards their severity. They may, for example, comprise merely a faint ciliary blush, trifling photophobia, and slight local discomfort, together with a discolored iris and a contracted and distorted pupil. Upon the other hand, redness may be marked and pain severe, the iris being greatly thickened and numerous posterior synechiæ being present. It is more important to note, however, that a certain proportion of cases manifest characteristic signs which permit the surgeon to identify the cause literally at a glance. To begin with, there are the so-called papules or condylomata,—that is, small raised masses embedded in the iris, and seldom exceeding two or three in number. They are most commonly met with in the pupillary zone, although they may be observed occasionally in other places,—for instance, the periphery of the anterior chamber. Commencing as small points, they increase in size, and very exceptionally may even become so large as to touch the posterior surface of the cornea; but, as a rule, their diameter is seldom more than two millimetres. While this is taking place, their color changes from a reddish brown to some lighter hue, and, upon using a magnifying glass, numerous fine vessels may be seen to surround them. Finally, they disappear completely, or leave behind them broad arc-shaped synechiæ or thinning and atrophy of the iris at the places where they were situated; exceptionally an actual hole in the iris marks the point where a papule formerly lay. These condylomata have a brief and transient life-history, so that we may accept them as occurring at some stage in the course of every case of specific iritis. A white line is not infrequently noticed at the bottom of the anterior chamber. This is due to the accumulated *débris* of broken-down condylomata, whether iridic or ciliary. A striking rusty hue of a localized portion of the pupillary edge of the iris is sometimes observed, and is due in all likelihood to condylomatous masses diffused throughout the tissues in that position. This seems to occur more commonly in the lower half of the pupillary edge than elsewhere. In the next place, the posterior synechiæ in this form of iritis are in some instances suggestive of the cause of the ailment. They are relatively broad, inextensible, and of dark color, thus differing markedly from those of rheumatic cases, which are usually fine, extensible, and of light color. Then, specific inflammation is not infrequently characterized by an abundant deposit of pigmentary material upon the anterior capsule of the crystalline lens. Lastly, there is an additional sign upon which stress was laid by

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the earlier writers,—a dislocation of the pupil upward and inward. Beer regarded this as characteristic of syphilitic iritis; but it may, in point of fact, be met with in other forms. Its consideration, therefore, need not detain us in this place.

(*b*) A form of irido-cyclitis is occasionally seen many years after venereal infection. It presents many points of analogy with the late iritis of hereditary syphilis, and, like that affection, is a tertiary manifestation. But, contrary to the rule, it is usually bilateral, and yields to a long course of mercury. When it occurs in association with other late disorders, as disseminated choroiditis, gummata of the muscles or viscera, periostitis, or ozæna, the recognition of its origin will be simple enough, but, in the absence of those signs, a causal diagnosis can be reached only from the history of the case. The affection is marked by dots upon the posterior surface of the cornea, by a vitreous haze, and by a liability to secondary glaucoma. There is but little tendency to the formation of posterior synechiæ. In some instances condylomata appear to have been present, if we may judge by the depressed, slaty-blue spots about the pupillary edge of the iris. Strictly speaking, these should be regarded, perhaps, not as condylomata, but as gummata. Arlt has made the important observation that, provided the pupil can be fully dilated, the latter may even be recognized in the flat portion of the ciliary body.

The following conclusions can be drawn concerning this form of disease:

1. It is seen, upon the average, about thirteen years after the primary sore.

2. It generally involves both eyes.

3. It takes the form of an irido-cyclitis, and is sometimes associated with the formation of gummata in the iris or the ciliary body.

4. It may exist along with other tertiary symptoms.

Rheumatism.—The qualifying adjective "rheumatic" has been applied by some writers to any iritis believed to be caused by cold. Apart from this loose use of the term, there can be no doubt that rheumatism (acute or chronic articular) is capable of predisposing to inflammation both of the iris and of the ciliary body. The evidence upon this point is clear enough. In the first place, a definite family or personal history of articular rheumatism may be made out in numerous cases of iritis for which no other cause can be found. Symptoms that are generally regarded as rheumatic—for example, lumbago, neuralgia, sciatica, torticollis, erythema nodosum, or pains in the articulations or fasciæ—are relatively common in these cases, and may alternate with swollen joints. The patients are remarkably sensitive to cold and to damp, while variations in weather are much felt by them. The relapses of iritis appear to be most frequent in winter and in spring,—*i.e.*, during the two seasons when rheumatic affections are acknowledged to be most prevalent. Then, in a well-marked group of cases iritis alternates with acute or subacute rheumatism; that is to say, when the eyes are affected the joints are comparatively well, and *vice versâ*. Lastly, in

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another group iritis recurs every time the joints become swollen. Therefore we conclude that rheumatism and the rheumatic diathesis are powerful predisposing causes of irido-cyclitis.

The inflammation may affect one or both eyes. It is especially prone to relapse, and, indeed, it is no exaggeration to say that recurrent iritis is generally due to rheumatism. Typical cases are characterized by marked photophobia, lacrymation, and ciliary redness. Pain, which is very liable to assume a paroxysmal type, is often so intense as to deprive a patient of rest either by night or by day. It shows a striking tendency to radiate into neighboring parts, such as the forehead, scalp, cheek, side of the nose, and upper gums. It sometimes reaches so exquisite a pitch that the patient dreads the least touch, and complains loudly that the pillow hurts him, that he cannot bear to have his hair brushed, and so forth. There is not much tendency to effusion of lymph, and hence the iris seldom shows any marked structural change. Condylomata, of course, are never observed. Posterior synechiæ, when present, are seldom pigmented. They are usually long, thin, and pointed, and show a decided disposition to stretch or to rupture when atropine is properly applied. Pigmentary deposits upon the anterior capsule of the lens are not, as a rule, met with. At the same time, a common and rather characteristic condition is for the pupillary border of the iris to be fringed with a narrow band of grayish lymph, which is generally incomplete at the upper part of the circle, and which thus roughly resembles a horseshoe in outline. The cornea may be bright and clear; or, on the other hand, its posterior surface may be "cross-hatched" or dotted over with fine opacities, although these changes, upon the whole, are not so frequent as in syphilitic iritis. Hypopyon may occur, but possesses no particular significance, while similar considerations apply to hyphæma. Some swelling of the free edge of the upper lid, slight chemosis, and tenderness of the globe may be present, and indicate that the ciliary body participates in the inflammation, although this occurs comparatively rarely. Lastly, it should be added that, notwithstanding numerous recurrences, sight may be but little affected.

There are two sub-groups the characters of which are sufficiently marked to warrant separate description. The first, which may be called "neuralgic iritis," begins with severe pain radiating into the various branches of the fifth nerve. Throughout the course of the disease pain forms a marked feature. It recurs with horrible violence towards nightfall, and is often out of all proportion to the objective signs of disease. Irido-cyclitis comes on in the eye corresponding to the side attacked by the pain. The second eye may become affected later in a similar way and under similar circumstances to the first. Most of these cases, it may be noted, recover completely. The second group includes those cases of iritis or cyclitis that appear to be directly excited by cold. Many of the patients date their eye-trouble from exposure, such as may be incurred by driving in the face of a cold wind or suddenly leaving a heated room. This form of disease may

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attack one or both eyes, and sometimes lasts for a few hours only. It is exceedingly prone to relapse under a renewal of the exciting cause.

TABLE CONTRASTING THE ORDINARY FORM OF RHEUMATIC AND SYPHILITIC IRITIS.

Rheumatic.	*Syphilitic.*
1. History of acute or chronic articular rheumatism, or of other so-called "rheumatic" symptoms.	1. History of acquired syphilis.
2. Pain, photophobia, and lacrymation marked.	2. Pain, photophobia, and lacrymation not equally well marked.
3. Condylomata of iris never observed.	3. Condylomata probably present in all cases.
4. Exudative changes slight.	4. Extensive exudative changes.
5. Posterior synechiæ often long, thin, and non-pigmented.	5. Posterior synechiæ usually short and deeply pigmented.
6. Iris often strikingly bright.	6. Iris often dull and "muddy"-looking.
7. "Cross-hatching" and punctata keratitis relatively uncommon.	7. "Cross-hatching" and punctata keratitis relatively common.
8. Comparatively little tendency to formation of pigment upon the anterior lens-capsule.	8. Marked tendency to formation of pigment upon the anterior lens-capsule.
9. Not associated with choroido-retinitis.	9. May be associated with choroido-retinitis, as evidenced by diseased spots in the choroid, blurring of the optic disk, etc.
10. Generally perfect recovery of sight.	10. Sight often impaired both during and after the attack.
11. Great tendency to relapse.	11. Much less tendency to relapse.

Gonorrhœa.—There appear to be two kinds of blennorrhagic iritis, of which one is a non-relapsing and the other a relapsing complaint.

The former is exceedingly rare. It is not associated with any changes in the joints or fasciæ, and comes on during the earlier stages of gonorrhœa. It may affect one or both eyes, and be accompanied by symptoms of severe inflammation. As a rule, there is free exudation, and sometimes the pupil is occupied by a considerable mass of bluish-gray lymph. If the case be taken in hand early, complete cure usually comes about.

The latter, on the contrary, is not infrequent. It is met with during the gleety stage of gonorrhœa, and is nearly always preceded by (or associated with) articular swelling and pain, the joints most often attacked being the knee, the hip, the ankle, the elbow, and those of the hand and foot. Pain about the foot, and especially of the plantar fascia and parts about the tendo Achillis, is often complained of. These articular and fascial conditions are of a chronic and relapsing character. Painful affections of the peripheral nerves, such as sciatica, seem now and then to be part of the affection, and may exceptionally replace the joint-changes. The patients frequently inherit a rheumatic or gouty disposition. They are often pale, thin, and weakly, and have in many instances suffered from acute or chronic rheumatism in childhood,—that is, before they were likely to have contracted a venereal disorder. They generally give an account of

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repeated attacks of gonorrhœa, to which they seem liable in more than ordinary degree. They often tell us that urethral discharge, lasting, perhaps, a few days only, follows every sexual congress, no matter how pure. There is a tendency with advancing years to outgrow, as it were, this remarkable diathesis.

The iritis seldom attacks both eyes at once, although both are liable to suffer sooner or later. Its onset is sometimes marked by few or many dotted deposits upon the posterior surface of the cornea, and this is followed by fine opacities in the vitreous humor and by a limited exudation of lymph from the tissues of the iris. In an equally large number of cases, however, inflammation is limited to the iris. Pain, photophobia, and lacrymation are not usually well marked or severe. A few of the cases seem to begin with symptoms of mild catarrhal ophthalmia, showing those of iritis or irido-cyclitis at a later stage only. This type of disease, as noted before, shows a marked tendency to relapse; but, although its course is essentially chronic and recurrent, many patients recover perfectly.

That the disease bears a definite relation to the gonorrhœa seems to be quite certain. It is not uncommon to find that the contagious malady is followed by (or associated with) joint-affections and then by iritis, a sequence of events that may be repeated many times. In some individuals the gonorrhœal rheumatism may alternate with the affection of the eye, the transition from one ailment to the other being strikingly rapid; the inflammation, as the patient puts it, "flies from eye to joint," and *vice versâ*. Then, again, each relapse or exacerbation of the irido-cyclitis may be associated with a renewal of the urethral discharge, even in the absence of fresh contagion. When all is said, we must admit, however, that these cases are closely allied with the ordinary rheumatic ones.

Gout.—The older writers described a disease which they termed "arthritic ophthalmia," and which they regarded as of gouty origin. Besides the ordinary signs of iritis, this ailment, according to them, presented three characteristic symptoms: (1) varicosity of the vessels, both of the bulbar conjunctiva and of the iris; (2) a peculiar white, frothy secretion from the mucous membrane of the eye; and (3) a narrow grayish-white ring—the so-called "arthritic ring"—surrounding the cornea either wholly or at its inner and its outer side only. Notwithstanding this sharply-cut clinical picture, comparatively few modern writers enumerate gout among the causes of iritis. Mr. Hutchinson, however, is of opinion that many cases of relapsing cyclitis are due to the inherited disease, although the patient himself may manifest no trace of podagra. Mr. Nettleship, again, speaks of gout as "apparently a cause of some cases of both acute and insidious chronic iritis."

There can be little doubt, however, that in rare instances gout may give rise to cyclitis, and more rarely still to iritis pure and simple. These affections are characterized by great obstinacy; and, although a majority of the cases recover permanently, some pass into a condition of chronic glaucoma

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and end in blindness. The rule is for one eye to suffer in the first instance and for the other to become involved at a later period in the history of the case. In their sudden onset, their rapid disappearance, and their liability to relapse, the symptoms resemble the arthritic attacks of acute gout.

Whilst admitting, therefore, the existence of a gouty cyclitis or iritis, we would strongly urge that no such diagnosis be made unless there be sufficient evidence to warrant it. Before it is arrived at, other probable causes of disease, as syphilis, rheumatism, or gonorrhœa, must be excluded. Then, the patient must give a definite family or personal history of podagra, or, failing that, must be the subject of actual gouty changes, such as characteristic deformity (or synovitis with deposits of urates) of his smaller joints, or tophi in the helix of the external ear, the cartilage of the eyelids, the periosteum, or the nose.

Diabetes.—The fact that those with diabetes mellitus are especially prone to iritis after cataract extraction has been long recognized by ophthalmic surgeons. In addition to this, however, Leber[1] has shown that there exists an irido-cyclitis which is directly due to diabetes. He observed among thirty-five diabetics with eye-affections nine instances of iritis. Wiesinger[2] has reported a series of similar cases. The disease generally assumes a plastic type, although hypopyon is not uncommon. Spongy exudations and hyphæma have been met with. Vitreous opacities, too, are now and again seen. Provided proper treatment be adopted, the inflammatory deposits readily undergo resorption. The prognosis, therefore, does not seem to be unfavorable. The conclusion from the foregoing facts is that a point should be made of examining the urine of every patient suffering from iritis.

Herpes.—Zona ophthalmica is a cause both of iritis and of cyclitis. Those affections may be primary or secondary to a keratitis bullosa. In the former case, inflammation usually comes on during the period when the vesicles are drying up. As a rule, it assumes a serous type; that is to say, it is marked by keratitis punctata, a deepened anterior chamber, a dilated or semi-dilated pupil, and heightened tension. Plastic or purulent iritis is rarely seen. The secondary form of irido-cyclitis either accompanies or follows herpes of the cornea. It may be met with even months after the latter has run its course. The prognosis of both forms appears to be favorable.

Malaria.—Irido-cyclitis may occur in those who have suffered from malarial disorders, such as ague, remittent, "Dutch," and "jungle" fevers. The affection of the eyes may come on many years after exposure to the predisposing cause. Its leading characteristics are two in number, namely, periodicity and tendency to relapse. A majority of the cases terminate in complete recovery, although the course of the malady is often most tedious.

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[1] Graefe's Archiv, xxxi. 4, S. 183.

[2] Ibidem, S. 203.

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Malarial irido-cyclitis is almost unknown in this country, except among those who have lived abroad in paludal districts.

Menopause.—Irido-cyclitis is sometimes observed in women at the "change of life." It is perhaps going too far to connect these two events as cause and effect, although the fact remains that no other etiological factor is to be discovered for the cases in question. It is possible, of course, that the alteration in general health so often observed at the climacteric period may be responsible for the eye-affection. The inflammation varies much in intensity. It may, for instance, last for a few weeks only, and be of a more or less trivial nature; or it may persist for an indefinite period, and terminate in secondary glaucoma. In most instances the cyclitic symptoms preponderate.

Meningitis.—A purulent inflammation of the ciliary body and other parts of the uveal tract is sometimes met with in young subjects suffering from meningitis, such as that which may follow disease of the middle ear. The inflammation, which may affect one or both eyes, is marked by iritis, hypopyon, and diminished tension. In case the fundus can be examined, opacities will be found in the vitreous humor, or the latter may be extensively permeated by inflammatory deposits, in which event a whitish or yellowish-white appearance may be noticed through the pupil. The inflammatory deposits have little or no tendency to perforate the globe. The case terminates in loss of sight and atrophy of the eyeball. The current notion attributes this remarkable affection to a direct extension of the meningeal inflammation to the eye along the optic nerve. Oeller believes, however, that it results from thrombosis of leucocytes in the vessels of the ciliary body. According to his view, this is followed by stasis and disintegration of the red blood-corpuscles, which, in their turn, form thrombi. Extensive diapedesis then takes place, and in this way the deposits in the vitreous are brought about.

When this affection ("pseudoglioma") is seen after acute symptoms have subsided, the diagnosis must be made from glioma of the retina. The following are the chief points of distinction between the two conditions: (*a*) *The history of the case.* In pseudoglioma an account is often obtained of an acute illness, probably marked by cerebral symptoms; whereas in glioma nothing of the kind will, as a rule, be forthcoming. (*b*) *The appearance of the iris.* In pseudoglioma the iris generally shows numerous posterior synechiæ, and its periphery is retracted. In glioma, on the contrary, adhesions are rarely present, while the iris is pushed bodily forward towards the posterior surface of the cornea. In other words, the anterior chamber is in glioma rendered uniformly shallow, but in pseudoglioma, although deepened at its periphery, its centre is shallow. (*c*) *Tension.* Tension is increased in glioma, diminished in pseudoglioma, except in its earliest stages.

It should, perhaps, be mentioned that there are two other conditions to which pseudoglioma presents some points of resemblance, namely, gross

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tubercle of the choroid, and those rare cases in which the fibro-vascular sheath of the crystalline lens fails to undergo absorption during intra-uterine life.[1]

Scrofula.—A form of relapsing irido-cyclitis has been described in young persons who present signs of scrofula,[2] past or present. It bears much resemblance to the late iritis of hereditary syphilis; indeed, it is possible that many of the cases described as "scrofulous" are in reality of syphilitic origin. However that may be, the disease is marked by keratitis punctata, discoloration of the iris, vitreous opacities, and fluctuations in tension. The very existence of a pure scrofulous irido-cyclitis, it should be added, is denied by more than one modern writer. There can be no doubt, however, that an iritis not infrequently accompanies the superficial vascular opacities of strumous keratitis.

Tubercle.—Whatever doubt there may be as to scrofulous iritis, there can be none with regard to the existence of a tuberculous irido-cyclitis. Cohnheim produced the last-named affection by inoculating the anterior chamber with morsels of tuberculous tissue, while more recently a similar result has been obtained by injecting a pure cultivation of the tubercle bacillus. Within three weeks or so after either experiment, inflammation of the iris was observed, and this was soon followed by an eruption of small grayish nodules upon the surface of that membrane. The growths became larger and more numerous, coalesced with one another, filled the anterior chamber, infiltrated the cornea, and ultimately burst through the eyeball. Most of the animals succumbed at a later stage to general tuberculosis, the result of infection from the eye.

In man, tuberculosis of iris or ciliary body may occur under two forms: first, as disseminated growths; secondly, as a solitary growth.

In the former, one eye is generally affected: from some published statistics (Hill Griffith), the left would appear to be more prone to the disease than the right. Some cases are preceded by a stage in which small reddish nodules develop on the iris, disappear spontaneously, and are later followed by an eruption of typical tubercular masses. Haensell and Leber believe that the primary growths are themselves tubercular in nature.[3] Ciliary redness is present, while tenderness of the globe is seldom absent. The iris becomes dull, thickened, and discolored, and few or many grayish points appear upon its surface, especially towards the periphery. Of course these vary in size according to the stage at which they are seen, but, speaking generally, they range from one millimetre to six millimetres in diameter.

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[1] Collins, E. Treacher, Royal London Ophthalmic Hospital Reports, vol. xiii. part iii.

[2] By "scrofula" we understand a by no means uncommon constitutional state denoted by a tendency to enlargement of the lymphatic glands, by certain relapsing inflammations of the skin and the mucous membranes, and in some instances by a characteristic facies and build of body. Pathologically, it is denoted by rapid growth and early caseous degeneration of the products of inflammation.

[3] Deutsche Medicinische Wochenschrift, October 1, 1892.

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As a rule, each is surrounded by a ring of minute blood-vessels, so that the base has a reddish hue. Individual tubercles, like the condylomata of specific iritis, run a short course and soon disappear, but are speedily replaced by a fresh crop. Broad synechiæ, posterior or anterior, often remain at the places where they formerly lay. A characteristic appearance is that of a single large grayish nodule surrounded by a number of small, translucent, satellite-like masses. In consequence of the irritation set up by the tubercles, the pupil may get "excluded" or "occluded," or grayish-white shreds of lymph may cover the anterior surface of the iris. The aqueous humor is always more or less cloudy, and it is not uncommon to find particles of disintegrated nodules lying at the bottom of the anterior chamber, where they form, as it were, a kind of spurious hypopyon. Hyphæma, too, may be observed. The cornea frequently shows local deposits of tubercle, which in their naked-eye characters resemble those found in the iris. Exceptionally the cornea becomes affected before the iris. Tension is not infrequently raised at this stage of the disease. In the further progress of these cases a plastic inflammation of the ciliary region is set up, which is followed by lowered tension and shrinking of the eye-ball, or the eye bulges in the ciliary region and perforation takes place. Death from miliary tuberculosis or tubercular meningitis is not uncommon at a later period. The local and general prognosis, therefore, is bad.

It is, however, of practical importance to recollect that in a certain number of instances the growths have become absorbed, the cornea has cleared, and the eye has to a great extent regained its natural functions. The patient, moreover, has remained in good health for years. It would be difficult to describe the clinical features of these cases of "attenuated tuberculosis" better than Leber has done. His words may accordingly be quoted: "The nodules, which are generally of the size of a millet-seed or less, are scattered in variable number over the iris. It is not uncommon for them to invade the pupillary border, there giving rise to a large synechia, or to occupy the angle of the anterior chamber, especially towards the periphery of the inferior sinus. Their color is yellow or yellowish gray. At a later stage vascularization may give to them and to the surrounding tissue a reddish tinge. Apart from the existence of these nodules, iritis or irido-cyclitis is present, and it may be either serous or adhesive in type. This is distinguished from the ordinary form of tuberculous iritis by its slow course, as well as by the smaller size and slower growth of the nodules. This augmentation in size at length comes to a stand-still, and a total regression of the nodules ends in a complete cure, while in other cases the malady continues for an indefinite time, with periods of amelioration and aggravation, of which one scarcely has the chance to see the termination."[1] "Attenuated tuberculosis" of the iris, one must bear in mind, may

[1] Bericht über die Versammlung der Ophthalmologischen Gesellschaft, Heidelberg, 1892.

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be followed by meningitis or phthisis and death, as in cases recorded by Samelsohn and by Hippel.

The solitary growth, the so-called *granuloma iridis,* begins as a round or oval nodule, which is of grayish-white color, and which in some instances arises from the lower part of the anterior chamber. It slowly increases in size, but without giving rise to marked inflammatory symptoms. After filling the anterior chamber, perforation takes place, with the result that a granulation-like, pale red or yellowish mass appears somewhere in the neighborhood of the sclero-corneal margin. Lastly, both the protuberance and the eyeball shrink, although a considerable time may elapse before this takes place. This exceedingly rare affection, which is unilateral, always entails, then, loss of the eye. It appears, however, to be at bottom the same thing as the disseminated form, inasmuch as tubercle bacilli have been demonstrated in both. Furthermore, inoculation of the eyes of rabbits with morsels of the growths has set up, in some instances, local and general tuberculosis.

Both the foregoing conditions occur in persons who, as a rule, are under twenty years of age and who have a family or personal history of tubercle. Many of the patients are phthisical, or present other signs of active disease, —for example, swollen or suppurating glands, joint- or bone-affections, or "strumous nodules" in the skin. There is still much difference in opinion as to whether the ocular disease is primary or secondary to some other tuberculous lesion.

From what has been said it will be evident, therefore, that the diagnosis of tubercular iritis will depend partly upon the anatomical characters and course of the inflammation and partly upon a family or personal history of tubercle in some of its various manifestations. In doubtful cases a characteristic nodule has been removed, so that it might be examined for bacilli or inoculated into the eye of a rabbit, but this method seems seldom to have yielded a positive result. In point of fact, tubercular growths are likely to be confused with four conditions only,—namely, (*a*) *syphilitic condylomata,* (*b*) *sarcomata,* (*c*) *ophthalmia nodosa,* and (*d*) *lepra nodules.* (*a*) *Condylomata* generally occur in persons over the age of puberty who give a history of syphilis or show signs of that malady. The growths seldom exceed two millimetres in size, are vascular, and disappear under mercurial treatment. (*b*) *Non-pigmented sarcomata* of the iris are single and very vascular. They get progressively larger, and never disappear spontaneously. It need scarcely be said that mercurials fail to influence them in any way. (*c*) *Ophthalmia nodosa*[1] is a curious and uncommon condition, of which about

[1] The following references may be serviceable to those interested in ophthalmia nodosa: Pagenstecher, Klinische Monatsblätter, xxi.; Weiss, Archiv für Augenheilkunde, xx., S. 341; Wagenmann, Archiv für Augenheilkunde, xxxvi., S. 126; Krüger, Archives of Ophthalmology, xxii., 1893; Becker, Berliner klinische Wochenschrift, May 30, 1892; Hillemanns, Deutsche Medicinische Wochenschrift, xxiv., S. 517; Lawford, Transactions of the Ophthalmological Society of the United Kingdom, vol. xv. p. 210; Elschnig, Klinische Monatsblätter für Augenheilkunde, 1895, S. 182.

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a dozen cases have been published. It is due to penetration into the eye of the hairs of certain kinds of caterpillars. It is marked by many small growths, not only in the iris but also in the lower part of the conjunctiva and sclera. It may run a chronic course, and entail violent and destructive irido-cyclitis. A history may be forthcoming of a caterpillar being thrown against the eye, or the patient may have been liable to such an injury by the nature of his occupation. Often all doubt as to the character of the affection may be laid to rest by excising one of the nodules and submitting it to microscopical examination, when it will be found to be made up of round and giant cells and to have a hair running through it. (*d*) *Lepra nodules.* An eruption of nodules upon the iris has been met with in leprous subjects. The condition, however, is seldom primary; it generally comes on as a complication of corneal or conjunctival leprosy. Should there be any difficulty in respect of diagnosis, a nodule might be excised and examined bacteriologically.

Traumatic Iritis, or *irido-cyclitis*, as well known, may follow various kinds of injuries. These may be divided into (*a*) mechanical and (*b*) chemical. The common mechanical injuries are contusions of the eyeball and penetrating wounds of the cornea, iris, ciliary body, or lens. The latter may be inflicted accidentally or be made by the knife of the surgeon, as in the operations of iridectomy and cataract extraction. Inflammatory symptoms may be slight and transient, or they may, on the other hand, be severe enough to cause loss of sight or sympathetic ophthalmitis. Thus, a contusion of the eyeball will probably entail nothing worse than a localized inflammation of the iris, while penetration of the globe by a septic instrument may cause purulent irido-cyclitis and destructive panophthalmitis. Although it would be beside our present purpose to enter into any discussion upon penetrating injuries of the eye, nevertheless it may be pointed out that the prognosis of such cases will mainly turn upon three factors. They are these: (1) the position of the wound with reference to the ciliary region; (2) the condition as to surgical cleanliness of the instrument by which the wound was inflicted; and (3) the presence or absence of a foreign body within the eyeball. A remarkable form of iritis is sometimes observed in detachment of the retina, in dislocation of the lens, and in tumors of the choroid. It seldom involves any marked inflammatory symptoms, but becomes manifest by some alteration in the color of the iris and by the presence of posterior synechiæ. It is in all likelihood the result of traction upon the tissues of the iris and ciliary body, and hence is of mechanical origin. Perhaps the best example of a chemical injury is furnished by the entrance into the eye of certain metals, as mercury and copper, which, according to Leber, have the power of causing purulent inflammation altogether apart from their septic or aseptic state. The irido-cyclitis (plastic) sometimes seen after cataract extraction is probably another case in point, although there can be no doubt that there mechanical irritation also comes into play.

Post-Febrile Iritis.—Irido-cyclitis has been observed to follow certain

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febricula, of which the most important are relapsing fever, small-pox, typhoid, and typhus. The disease usually takes the form of a serous cyclitis, with dots upon the posterior surface of the cornea, and a haze in the vitreous humor. Whilst running a lingering course, it generally ends in cure. Purulent irido-cyclitis due to septic embolism may occur in septicæmia after childbirth or surgical operation. The affection is unilateral, and the eye is always lost.

Cachectic.—Irido-cyclitis (or iritis) is sometimes met with in those debilitated by recent illness,—for example, pneumonia or influenza (Laqueur, Adler, Eversbusch, Natansen). It is occasionally set up by "over-lactation." This group appears to be sufficiently well marked to warrant separation from the post-febrile cases, on the one hand, and from the idiopathic, on the other. The inflammation, however, presents no characteristic features, except, perhaps, a tendency to insidiousness.

Idiopathic Iritis.—Many other causes besides those enumerated in the preceding pages have been assigned for iritis,—for instance, Ménière's disease (Knapp), varicella (Steffan), cholera (Williams), albuminuria (Higgens, Knies), mumps (Schiess), leucæmia (Michel), erysipelas (Cornwell), stricture of the urethra (Despagnet, Brun), favus (Rampoldi), psoriasis (Morax), uterine disorders (Cohn, Trousseau, Wecker, Grandclément), nasal troubles (Ziem, Fage, Berger), and dental affections (Faucheron, Brunschvig). There are, however, not a few cases in which we fail to recognize the cause, and for the sake of convenience such may be grouped together under the common name "idiopathic." No surgeon, of course, would apply this title until he had inquired into the family and personal history, examined the patient's body for signs of syphilis, rheumatism, gout, and gonorrhœa, and tested the urine; the fundus of the eye, we may suppose, would also be explored. In short, before deciding to call any case idiopathic, every known cause of iritis and cyclitis would, so far as possible, be excluded.

General Remarks upon the Pathogenesis of Primary Iritis.—In glancing over the preceding pages one can scarcely fail to note that almost all the ailments capable of giving rise to inflammation of the iris are intimately associated with, if not actually caused by, micro-organisms. As regards syphilis, gonorrhœa, tubercle, leprosy, influenza, and relapsing fever, this fact will be disputed by none. With regard to rheumatism, the case is by no means so clear, although the view is gaining ground that it also is due to specific infection. Hueter seems to have been the first to advance this theory. Newsholme,[1] as the outcome of an extensive statistical study of the disease, has recently stated that acute articular rheumatism must be included among the infective diseases. He has been supported in this view by other observers,—for example, Knies[2] and F. R. Humphreys.[3] If the

[1] The Lancet, March 9 and March 16, 1895.
[2] Relations of the Diseases of the Eye to General Diseases, New York, 1895, p. 374.
[3] Medical Press and Circular, November 6, 1895.

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bacterial origin of acute rheumatism be admitted, its truth as applied to the chronic form of arthritis can hardly be denied. Lastly, organisms have been described in many fevers,—typhoid, small-pox, and pneumonia,—during recovery from which inflammatory affections of the iris and ciliary body may occur.

Two propositions, then, appear to be clear: first, that most inflammatory affections of the iris and ciliary body are dependent upon a constitutional ailment; secondly, that a majority of those ailments are of a bacterial nature. The conclusion follows that most forms of irido-cyclitis result from the action of micro-organisms. It is possible that the immediate cause of these inflammations is to be sought in the vicarious glandular excretion of microbes (or their products) circulating in the blood or other nutrient fluids of the body.

More than thirty years have elapsed since Memorsky[1] proved that chemicals circulating in the blood might find their way into the aqueous humor. He injected a solution of potassium ferrocyanide into the veins, and twenty minutes later was able to demonstrate its existence in the aqueous humor by the perchloride of iron reaction. More recently, Ehrlich[2] noted coloration of that fluid after fluorescine had been introduced beneath the skin. These observations prove, therefore, that soluble salts, when thrown into the circulation, may be excreted by the iris and ciliary body. Evidence will be next adduced to show that micro-organisms may be thus got rid of.

The following, reported by Gillet de Grandmont,[3] is a case in point. That surgeon was consulted by a young man convalescent from typhoid fever. The patient was thin and weak, had albumen in the urine, and a jaundiced hue of the skin. His left eye was affected with iritis; it showed numerous posterior synechiæ and hypopyon, and its vision was so impaired that fingers could be counted with difficulty at the distance of one metre only. Grandmont opened the anterior chamber of the inflamed eye and inoculated a tube of agar-agar with some of the pus. Two days later he obtained a pure culture, which when examined with the microscope was found to be made up of the bacilli described by Eberth as present in the spleen, glands, and Peyer's patches of those with typhoid fever. A small quantity of this culture was next injected into the vitreous humor of a rabbit, which when killed three weeks afterwards was found to have numerous typhoid bacilli in the liver and intestines.

The same author[4] has found streptococci in a culture obtained by inoculating gelatin with the aqueous humor from a case of hyaloiditis (or, rather, cyclitis) following erysipelas.

Other instances may be quoted in which micro-organisms have been

[1] Archiv für Augenheilkunde, 1865.
[2] Deutsche Medicinische Wochenschrift, 1882.
[3] Archives d'Ophtalmologie, 1892, p. 623.
[4] Ibidem.

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demonstrated in the anterior chamber. Herman Snellen,[1] for example, has lately reported the following cases: (1) A lady, aged twenty-eight years, had marked descemetitis of the left eye, the lower third of the cornea being speckled over with punctate opacities. Increased tension coming on, sclerotomy was performed. One of the punctate dots escaped with the aqueous humor, and was found to be made up of very short bacilli, capable of cultivation on agar-agar. This patient later developed descemetitis of the other eye, in one of the dots from which identical microbes were discovered. (2) A young man had a similar affection of his right eye. The anterior chamber was tapped, a dot examined with the microscope, and found to consist of cells and short bacilli. Snellen concludes from these facts that descemetitis is a disease *sui generis*, and that "it is due to microbes growing in the anterior chamber, which by their producing toxines cause an irritation of the uveal tract." The pathogenesis of his cases would perhaps be better explained by assuming that both were due to some constitutional condition of microbic nature, and that the organisms found in the anterior chamber represented an excretion of the virus by the ciliary body.

Blennorrhagic iritis arises at the same period as blennorrhagic arthritis; that is to say, after the symptoms of acute gonorrhœa have yielded to a gleety discharge from the urethra. Now, it is a significant fact that inflammation of other serous membranes has been noted as arising during the same period. Mackenzie[2] many years ago mentioned the case of one patient in whom the virus seemed to produce chronic peritonitis, and of another who died from inflammation of all the internal serous membranes. Mazza saw pleurisy, peri- and endocarditis, and polyarthritis during an attack of gonorrhœa. In the effusion from the pleural cavity he found Neisser's gonococcus. Bordoni-Uffreduzzi[3] has described multiple arthritis during gonorrhœa. Fluid taken from a joint was found to contain the characteristic organisms, which after cultivation were inoculated into the urethra of a young and healthy man who had abstained from coitus for some months: a typical attack of gonorrhœa was the result, and gonococci were demonstrated in the discharge. So far the genital tract has been the starting-point of these various inflammations, but that is by no means essential. In 1881, Poncet[4] noted swelling of the knees and other joints after gonorrhœal pus had been applied to an eye with pannus. Four years later it was shown by Lucas[5] that arthritis might develop in infants with purulent ophthalmia. He narrated three instances of the kind. His observations have been confirmed by Fendick, Debierre, Saswornitzky, Darier, and others. Deutschmann[6] noticed acute inflammation of the knee in a child

[1] Ophthalmic Review, 1894, p. 259.
[2] Practical Treatise on the Diseases of the Eye, London, 1854, p. 555.
[3] Archives italiennes de Biologie, 1895, t. xxii. p. cliv.
[4] Archives d'Ophtalmologie, 1881, t. i. p. 213.
[5] British Medical Journal, February 28, July 11, and October 10, 1885.
[6] Archiv für Ophthalmologie, xxxvi., 1, S. 109.

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three weeks after purulent ophthalmia. He found typical gonococci, not only in the conjunctival secretion, but also in the fluid obtained from the knee-joint by puncture. Haushalter[1] has recently reported a similar case. These facts appear to admit of but one explanation,—namely, that under certain circumstances the gonococcus is able to find its way into the blood, from which it is excreted by the synovial and serous membranes. This vicarious excretion sets up a corresponding inflammation.

The facts at disposal appear to indicate that syphilitic iritis is due to the local presence of microbes.[2] Thus the ordinary form of disease appears as an early secondary symptom during the time when the specific virus is diffusing itself by means of the blood throughout the entire economy. Nodular growths, or condylomata, are probably present in every case, and are strongly suggestive of local irritation, such as might very well be set up by bacilli deposited from the aqueous humor. It is a striking fact that the irido-cyclitis of hereditary syphilis may be preceded by, or associated with, arthritis of the knee or other large joints.

Some recent experiments performed by Ahlström[3] certainly appear to favor the view that rheumatic iritis is an infectious malady. He has succeeded in setting up iritis in rabbits by inoculating the anterior chamber with morsels of iris taken from a case of rheumatic inflammation. No such result was observed when the iris was derived from a non-septic source, such as an eye with uncomplicated cataract.

Lastly, many of those with irido-cyclitis tell us that the disease was set up by some slight injury to the eye, or by exposure to cold, or by strain. Bronner[4] has given particulars of three cases in which concussion of the eyeball was speedily followed by local syphilitic disease. The same sort of thing is not unknown in acute pneumonia and in some other infectious maladies. So far as the eye is concerned, these injuries, by increasing the vascular supply, may perhaps be assumed to enhance the functional activity of the glandular apparatus, and hence to determine the excretion of organisms or their products.

Secondary Iritis.—A secondary iritis or irido-cyclitis may be met with in the course of many inflammatory affections of the eye, especially when the tissue primarily involved is contiguous to the anterior part of the uveal tract. For example, those affections are not infrequently found along with mycotic and septic ulcers of the cornea; scleritis and sclerosing keratitis, again, are often associated with them.

Sympathetic Iritis.—Nothing need be said here with regard to this important disease, since it is described in another part of this volume.

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[1] La Semaine Médicale, 1895, t. xv. p. 382.

[2] Alexander believes that specific iritis is produced by vascular alterations. In support of this view, he points to the fact that Fuchs and Friedel have shown that the walls of the vessels of the iris are the seat of a gummatous degeneration.

[3] Beiträge zur Augenheilkunde, October, 1895.

[4] Transactions of the Ophthalmological Society of the United Kingdom, vol. x. p. 199.

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TREATMENT OF IRITIS AND CYCLITIS.

The treatment of iritis and cyclitis may, broadly speaking, be said to run on similar lines, so that a common description will suffice. In each affection the main indications are threefold: 1. To dilate the pupil and to keep it dilated until inflammation has subsided. 2. To relieve pain, congestion, and photophobia, to secure rest for the inflamed eye, and to combat sleeplessness. 3. To treat any constitutional ailment that may lie at the root of the mischief.

Unless there be reasons to the contrary (see pages 305, 306), dilatation of the pupil is the first point to be aimed at by the practitioner. This is needed for several reasons. It carries the inner margin of the iris away from the axis of the pupil, thus reducing to a minimum the chances of adhesion between the two structures. It is well to bear in mind, however, that with even the widest possible dilatation of the pupil it is possible for synechiæ to develop, although in this latter case they will lie well away from the central region. It may be noted in passing that the appearance produced by such an adhesion will eventually resemble that following the performance of a narrow iridectomy. To resume: soft and recent synechiæ may be often stretched, if not actually ruptured, by a wide and vigorous dilatation. Again, the mydriatics commonly employed paralyze the ciliary muscle, and as a consequence the eye is placed at rest. Lastly, it is probable that atropine (the agent generally selected) acts as a direct sedative upon the inflamed iris, while it certainly contracts the vessels of that membrane, and thereby hinders exudation.

Pain, photophobia, congestion, and other evidences of inflammation will to a great extent be relieved by full dilatation of the pupil, so that in slight cases nothing more need be done. As matters improve, the mydriatic is used less frequently, and finally discontinued altogether.

Mydriasis, as a rule, is secured by atropine, a medicament that may be used in various ways and under various forms. The every-day plan is to employ a solution of the sulphate containing two to four grains to the ounce of distilled water, one drop or more of this liquid being put into the eye three to six times a day, according to the severity of the inflammation. It is a good plan, however, in recent iritis, to direct that an application be made every ten minutes for one hour, as a cumulative effect may perhaps be obtained more easily in that way than in any other. The pure sulphate and not the alkaloid should be dispensed, since the latter is only slightly soluble in water, and the acid or spirit sometimes added by the chemist with the idea of increasing its solubility often acts as an irritant upon the eye. It is advisable to prepare small quantities of the solution only, so that the chance of fungoid growth may be lessened; for a similar reason many surgeons use camphor water as the menstruum, or add a minute proportion of some antiseptic to the distilled water. The analgesic effects of atropine may be enhanced by the addition of the hydrochlorate of

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cocaine, in the proportion of four to eight grains to every ounce of the foregoing solution. Moreover, the mydriasis produced by a mixture of the two salts has been found to be greater than can be obtained from either singly.

Atropine may be also employed in the form of ointment, but only when lacrymation is not excessive. The surgeon must remember that the salts of atropine are not themselves soluble in vaseline, whereas the alkaloid is. The former, therefore, should not be prescribed. The following is a good formula: atropine and cocaine, of each one part; vaseline, one hundred parts; dissolve by the aid of gentle heat. The ointment, which may be used three or four times in the twenty-four hours, may be applied to the eye in various ways, a simple plan being to spread a small amount upon lint, which is then bandaged into position. Perhaps, however, a more certain and convenient way is to insert the remedy between the lower lid and the eyeball by means of a small camel's-hair brush previously charged with the medicament.

In a third method—recommended by certain German authors—a morsel of the solid sulphate (*not the alkaloid*) is placed in the conjunctival sac and allowed to dissolve *in situ*. As alarming constitutional symptoms are not uncommon, this application should be made by the surgeon himself, while for a similar reason it should be reserved for cases of special difficulty. There are two conditions in which the solid sulphate sometimes renders signal service: the first, when the pupil obstinately refuses to dilate, notwithstanding the careful use of the ordinary preparations; the second, when one desires to rupture recent synechiæ.

There are instances where the use of atropine in any form gives rise to the group of symptoms collectively known under the name of "atropinism." This may manifest itself in either a local or a general way. The local form of atropinism is characterized by redness and swelling of the conjunctiva, along with the development of follicles in that membrane, and the presence of a muco-purulent discharge from the eye. At other times one may meet with a condition that bears a close resemblance to erysipelas of the face, the tegument of the eyelids and neighboring parts becoming irritable, swollen, and red. The general signs of atropine intoxication comprise clamminess of the mouth and dryness of the fauces, flushing of the skin, thirst and dysphagia, hallucinations and delirium. The frequency of the pulse is always increased. As a further result, retention of urine may take place, although that appears to be seldom met with except in aged persons. Even death is stated, upon the authority of Kugel, to have resulted from the local application of atropine.

The urgent symptoms of atropine poisoning may be combated by injecting beneath the skin one-fourth of a grain or more of morphine, as well as by the liberal administration of strong coffee. Atropine must, of course, be at once withdrawn, and recourse had to other mydriatic agents, such as daturine, duboisine, hyoscyamine, scopolamine, or homatropine. As a

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matter of fact, however, there exist only three pure solanaceous alkaloids,—atropine, hyoscine, and hyoscyamine,—for it is now recognized that daturine is nothing more than pure atropine, while duboisine is the same thing as hyoscyamine. Be that as it may, under the circumstances detailed above, surgeons generally employ the sulphate of daturine under the form of an aqueous solution containing one to two grains of the salt to one ounce of distilled water. Even daturine may give rise to toxic symptoms, and in that case the surgeon may substitute for it the sulphate of duboisine in a solution containing one to two grains to the ounce. Hyoscyamine is generally employed in this country as a small gelatin disk containing $\frac{1}{5000}$ grain of the alkaloid. The action of this agent is more potent than that of atropine, and its effects last about as long. Scopolamine has been used as an aqueous solution containing one grain of the drug dissolved in one ounce of distilled water. The salts of homatropine (hydrobromate, hydrochlorate, and salicylate) are readily soluble in water, and a common strength is eight to ten grains to the ounce. As the effects of homatropine, however, speedily pass away, the agent is inferior to those just described.

The local symptoms of atropinism are to be met by immediately stopping the irritating agent and by the application of astringent lotions and ointments to the inflamed skin. Among the latter may be mentioned the excellent unguentum metallorum, which combines equal parts of acetate of lead, oxide of zinc, and dilute nitrate of mercury ointments.

The surgeon should carefully note that there are, altogether apart from "atropinism," certain conditions in which atropine does more harm than good. For instance, it is by no means rare to come across cases of cyclitis (or irido-cyclitis) where the employment of that agent intensifies any pain, photophobia, or congestion that may be present. It has been suggested that this is due to a damming back of blood in the ciliary body as the result of the constriction of the vessels of the iris which is necessarily associated with mydriasis. The practical point is that under circumstances of this kind we must content ourselves with a partial dilatation of the pupil, such as may be obtained by using a feeble solution of atropine sulphate (one-twelfth to one-sixtieth of a grain to the ounce of water). Heightened tension of the eyeball, so often observed during the earlier stages of cyclitis, furnishes a second contraindication to the use of atropine. When this is the case, an attempt must be made to lower tension by the application of myotics, and for this purpose the sulphate of physostigmine is generally selected. It would be unwise, however, to employ anything like a strong solution of that alkaloid, because any exudation that might be thrown out would then be likely to encroach upon the pupillary area, thereby compromising sight seriously. It would be better to trust to weak physostigmine (one-fourth to one-half grain to the ounce), alternating it, if necessary, with an excessively attenuated solution of atropine. Cocaine, used either alone or combined with physostigmine or pilocarpine, might succeed in some of these difficult cases. The systematic employment of hot poppy

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fomentations has some effect in lowering tension, and purgatives seem to act in a similar way, so that these accessory measures should not be neglected. In case these various remedies fail, it will be necessary to open the anterior chamber of the eye, or, as a last resource, to proceed to iridectomy. But more precise indications for the performance of these surgical operations will be given later.

Heat and cold are useful in relieving pain and in modifying inflammation. It is difficult to formulate any rule as to which should be employed in a given case, but the patient's sensations form, perhaps, the most trustworthy guide in that respect.

Heat may be employed either in moist or in dry form. Poultices were formerly much in vogue as a means of applying moist heat to an inflamed eye, but nowadays they have been largely replaced by more cleanly appliances. The following is an excellent plan. Several folds of lint are dipped into hot water, squeezed as dry as possible, applied to the lids, and covered with oiled silk. This dressing is then packed in with a thick layer of dry wool heated by contact with the outside of a can of boiling water, and bandaged into position. A more simple but equally efficacious plan directs that a napkin be wrung out of hot water and rapidly applied to the closed eyelids. The first napkin is replaced by a second as soon as the former has lost its heat. There can be no objection, from a medical point of view, to using hot infusion of chamomile flowers or poppy fomentations instead of plain water. Sir William Bowman was in the habit of employing conium lotion, made by adding one drachm of the extract to each pint of water; while belladonna fomentations (extract of belladonna, one drachm to a pint of water) still enjoy great repute with many practitioners. Hot applications are generally ordered for half an hour twice or thrice a day, but their precise number will depend upon the nature of the case, and especially upon the amount of pain that may be present. There is one small point that calls for passing mention,—namely, that a delicate skin may become fretted by the moisture. It is, then, a good routine practice to smear the lids and neighboring parts with vaseline before putting on the hot applications.

Dry heat is most conveniently obtained by covering the eyelids with a large pad of cotton wool previously heated by contact with the outside of a can containing boiling water. Another plan is to warm the bunch of wool by holding it in front of the fire or by placing it in the oven.

Alternate use of moist and of dry heat is recommended by some authorities, although it is difficult to understand upon what grounds.

Cold may be applied in three different ways. In the first plan a piece of tape (one inch broad) is bound around the patient's head, and to it is pinned a square of linen, which covers the inflamed eye. A lump of ice floats in a basin containing water, with which the rag is kept constantly moistened. The temperature of the fluid is in this way maintained at a low point. The second plan may be thus described: by the side of the bed is

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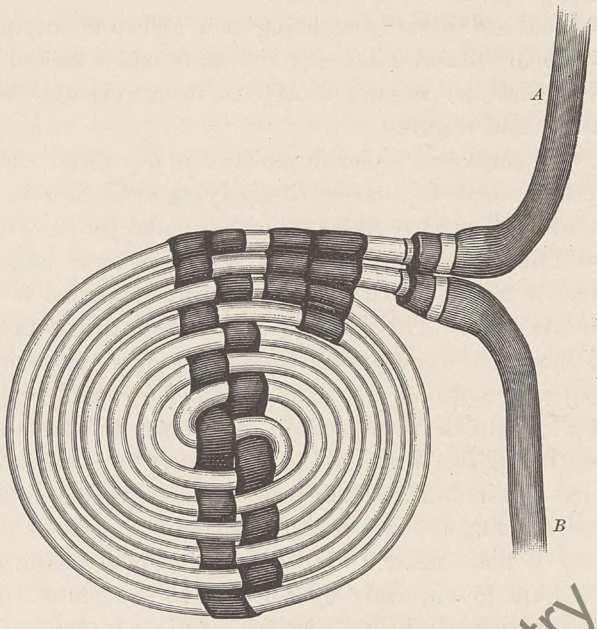
placed a large block of ice; two pads of cotton wool are provided, of which one is laid upon the ice and the other upon the eye of the patient, and they are changed as often as the one in use ceases to give a sensation of cold. The third plan is to employ a modification of the familiar Leiter's tubes,

Fig. 6.



Leiter's tube for applying cold to the eye.

which, as ordinarily constructed, are far too heavy to be used in eye-work. Fine leaden tubing, as shown in the diagram, is coiled into a disk of about two inches' diameter, the individual strands being held in place by narrow tape bands, which are stitched together. The contrivance is then placed in contact with the closed eyelids of the patient, who must be lying down. One free end (*A*) is brought by means of elastic tubing into connection with a vessel filled with iced water and suspended at some height above the bed. The other end (*B*) of the leaden piping is similarly connected with a receptacle placed upon the floor. The upper tube being now filled with water, siphon action will insure a constant flow of liquid through the coil until the reservoir is exhausted. This little apparatus, obviously, may be also used to apply heat to an eye.

Local abstraction of blood—a time-honored remedy in iritis—is sometimes a useful accessory to the employment of mydriatics. It is specially indicated when pain or congestion is marked, or when the pupil refuses to respond to the intelligent and correct application of atropine. It may be carried out in two ways,—by the use of leeches, natural or artificial, or by the operation of arteriotomy.

If leeches be selected, three or four may be applied to the temple, the

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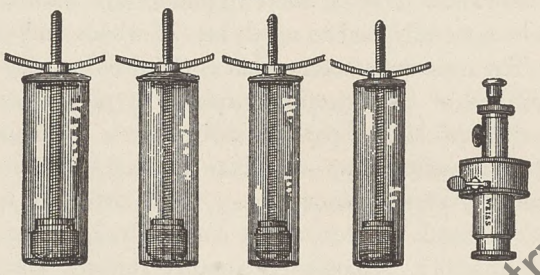
forehead, the side of the nose, or behind the ear, the application being repeated a second, third, or fourth time at intervals of a day or so. The skin is first washed, and the creature held in contact with it by means of a test-tube. If the animal will not bite, the part selected for its operations may be smeared with cream or with a little fresh blood. Each leech abstracts about a drachm and a half of blood, and will drop off when full. After leeching, certain complications may be met with, particularly in those whose general health is depressed. Thus, the wounds may bleed excessively, or septic inflammation may be set up. The space at disposal, however, will scarcely admit of our detailing the management of these untoward results, full particulars of which may be found in any book on general surgery. Lastly, it is often a good plan in the severer cases of iritis to encourage bleeding by warm fomentations continued for an hour or more after the leech has fallen off.

Heurteloup's "artificial leech" was formerly in great vogue. As shown by the figure, it consists of two parts,—a sharp drill and a cupping glass

FIG. 7.



Heurteloup's "artificial leech."

provided with a screw-piston. The former is driven into the skin of the temple, and blood is drawn away by exhausting the glass. This is, at least, the theory of the instrument, although in practice the piston generally refuses to act properly. It is better, therefore, to employ a slight modification of the original apparatus, in which the glass receptacle is exhausted by means of a small air-pump, kept constantly working by an assistant during the operation. The proportion of cases benefited by the "artificial leech" is small, even supposing that it works efficiently, so that its use is becoming more and more restricted. Possibly it may be of service in strong and plethoric subjects, but it should certainly never be used in young persons or in those debilitated from any cause.

Arteriotomy, albeit a speedy means of reducing inflammatory symptoms in suitable cases of iritis, has almost completely dropped out of fashion, and is seldom heard of nowadays. It is, however, a better and more scientific way of abstracting blood than that just mentioned. The operation is simple. It consists in nicking open the anterior branch of the temporal artery as it crosses the temple to its ultimate distribution. When enough blood has been abstracted, the vessel, hitherto only partially divided, is cut

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completely across, so as to avoid any risk of consecutive aneurism. A pad and bandage are then applied, in order to prevent further hemorrhage.

Counter-irritation is most serviceable, perhaps, in rheumatic iritis and in chronic irido-cyclitis. The more chronic the affection the more likely will it be to do good. The usual plan is to paint a limited portion of the skin of the temple with the liquor epispasticus, and to repeat the operation daily, selecting, however, a fresh spot upon each occasion. Instead of blistering fluid, one may use the tinctura iodi decolorata, which contains iodine (two hundred and fifty grains), rectified spirit (five and a half ounces), and strong solution of ammonia (ten drachms). An old and not inefficient method of counter-irritation is to rub lightly the cutaneous surface of the upper lid with a slightly moistened stick of pure lunar caustic. The œdema set up by this agent, however, renders the subsequent application of remedies to the eye rather difficult. Lastly, chronic cases have been treated by applying the actual cautery behind the ears or to limited portions of the skin of the temple.

Should pain be a marked feature, then special measures must be adopted to relieve it. For this purpose mere hypnotics are seldom adequate, and recourse must be generally had to anodynes, of which undoubtedly the best is morphine. The most satisfactory plan is to inject beneath the skin of the temple two minims of the injectio morphinæ hypodermica, repeating the dose a couple of hours later if pain be still present. This operation is best carried out late in the afternoon,—that is to say, before the nocturnal exacerbation, so commonly present, comes on. A full dose (ten to fifteen grains) of Dover's powder (pulvis ipecacuanhæ compositus), administered at night, will often act admirably. Aconite is another remedy which has its advocates. One drop of the tincture, suspended in water, may be given hourly until relief is obtained, or large doses (one-half to one and one-half grains) of the extract may be administered less frequently,—say, twice or thrice daily. Phenazonum (or "antipyrin," as it is termed), in doses of five to fifteen grains, twice or thrice a day, may sometimes succeed, and so may acetanilidum (or "antifebrin"), given up to twenty grains in the twenty-four hours. Fifteen-grain doses of "hypnal" (a compound of chloral and antipyrin) have been found of service. Unguents containing anodynes have been recommended. One of the oldest and most trustworthy combines two grains of powdered opium with ten grains of the strong mercurial ointment, and is directed to be rubbed into the temple before the advent of the nocturnal pain. The æsthetic objections now and then raised to this ancient preparation may be met by using a mixture of the oleates of mercury, atropine, and aconitine in a precisely similar way.

There can be little doubt that the action of mydriatics is materially enhanced, pain relieved, and inflammatory symptoms shortened by profuse perspiration. A good deal of importance is accordingly attached by most writers to a proper action of the skin. This may be secured by a succession of hot baths, taken the last thing at night, or by a course of Turkish

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baths, although the latter will probably be available only for those who are able to leave the house. The most certain means of inducing diaphoresis, however, is by the use of jaborandi, or rather of its active principle, pilocarpine. The usual plan is to inject subcutaneously one-twelfth to one-third grain of the nitrate salt, repeating the process every other day or at shorter intervals. Five-grain doses of Dover's powder, three or four times a day, besides relieving pain and sleeplessness, exert a marked action upon the skin in some cases.

A word with respect to "specifics" for iritis, of which more than one has from time to time enjoyed a wide vogue. It was formerly the custom to administer mercury in every case. "Its influence," wrote Lawrence,[1] "is not confined to the syphilitic form of the disease, but extends equally to the idiopathic" (as opposed to syphilitic?). Hence patients were indiscriminately saturated with the drug, which was believed to have the power of causing resorption of any lymph that might be effused in or about the pupil. This pernicious practice, fortunately, is all but given up at the present time. By the majority of surgeons mercury is almost entirely reserved for cases intimately associated with, if not actually caused by, syphilis. In fact, the only exception to this general statement is constituted by sympathetic ophthalmitis, an affection in which there appears to be actual evidence as to the value of mercurial preparations. The oil of turpentine,[2] too, has been lauded as a specific for iritis; but, like mercury, it also has fallen upon evil days, and its use is now more honored in the breach than in the observance. Similar remarks apply equally to balsam of copaiba (Hall) and to other agents which need not be specified.

Before we leave the symptomatic treatment of iritis and cyclitis, something may be said with regard to the general management of those maladies. In the first place, it is essential to secure rest for the inflamed eye, which may be done in several ways. In severe cases the patient may very well be confined to the house, at any rate until the more acute symptoms have been subdued. It is never necessary, however, to resort to that barbarous relic, the darkened room, which is not only irrational in principle, but may be actually harmful in practice. In cases of less severity protection may be secured by means of a double shade large enough to cut off all side light, although greater comfort is sometimes obtained by keeping the eye bandaged. In chronic cases the eye should be screened from light, wind, and other irritating agencies by smoked glasses or by domed protective goggles. The latter are seldom ground with any approach to accuracy, so that, upon the whole, flat glasses are preferable. It need scarcely be added that the patient must on no account attempt to use his eyes until the last trace of inflammation has died away. The neglect of this self-evident precaution is in all likelihood responsible for many relapses.

[1] A Treatise on the Diseases of the Eye, London, 1844, 3d ed.

[2] Carmichael, Observations on the Efficacy of Turpentine in the Venereal and other Deep-seated Inflammations of the Eye, etc., Dublin, 1829.

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As to diet, it is advisable that the patient should be restricted to milk, chicken broth, beef tea, and other light, digestible food during the height of an iritis or other acute inflammation of the uveal tract. As soon as the severity of the symptoms abates he may, however, resume his every-day diet, supposing that to be sufficient in quantity and quality. Stimulants, as such, are seldom needed, although it is obviously unwise to interfere actively with a patient's usual habit of life.

It need hardly be said that a patient should be warmly clad, and that great care should be taken to avoid cold and wet.

It is advisable to commence the treatment of every acute case by the administration of a purgative. Nothing is, perhaps, better for this purpose than the old-fashioned blue pill (five grains) and black draught (one-half to one and one-half ounces), although, if thought desirable, the former may be replaced by calomel (one to three grains), and the latter by Hunyádi water or by a Seidlitz powder. After this preliminary purge a daily action of the bowels may, if necessary, be secured by a teaspoonful of pulv. jalapæ comp., or by a similar quantity of pulv. glycyrrhizæ comp.

Those who suffer from relapsing inflammations of the iris and ciliary body will do well to conform in their mode of life with certain rules which are sanctioned no less by experience than by common sense. They should, for instance, be warmly clad, avoid extremes of heat and cold, and sleep with the head well raised by pillows. They ought not to keep late hours, while hot and overcrowded rooms should be sedulously shunned. Neither should they overtax the eyes by prolonged reading, sewing, and so forth. It goes without saying that they should live temperately, both as regards food and alcohol. Any constitutional ailment, especially rheumatism, should be carefully treated. If patients are in a position to afford it, a change to a warm, equable climate, or a periodical visit to a British or Continental spa, may suffice to ward off attacks. It stands to reason that prompt attention should be paid to any symptoms indicative of a recurrence, and it is perhaps worth while to remark that in some instances purgatives seem to be useful in this connection.

We may now pass forward to consider the causal indications of iritis and cyclitis,—*i.e.*, the treatment of any constitutional ailment that may lie at the root of those affections. In this connection it is but natural to accord the first place to syphilis, which, as we have already pointed out, is certainly responsible for at least one-half of the cases. Mercury under these circumstances is the remedy *par excellence*. In order to prevent disastrous consequences to sight, its exhibition should be promptly commenced, and it is a point of practical importance to obtain its physiological effects as speedily as possible. There are three ways in which this may be done,—viz., by inunction, by the vapor-bath, and by hypodermic injection. Inunction—the method commonly employed in this country—is certainly one of the simplest and best. It merely consists in rubbing into the skin from thirty to sixty grains of mercurial ointment, the patient having pre-

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viously taken a warm bath. The usual plan is to rub the medicament in at night only, but in urgent cases it may be done in the morning as well. The application may be made to the skin of the belly, of the axilla, of the thigh, of the arm, and of the temple, but a different point should be selected upon every occasion, as eruptions may otherwise be set up. In the case of infants it is sufficient to place the drug upon a strip of flannel, which is then bound around the waist. It may be added that if the unsightly appearance of the blue ointment be objected to, the oleate of mercury (ten per cent.) may be used instead. Without entering into unnecessary details, it may be said that the vapor-bath consists of a simple apparatus whereby water and calomel are volatilized and deposited upon the patient's skin. The bath is usually taken once a day, thirty grains or more of calomel being used upon each occasion. The system becomes affected, as a rule, within three or four days. Although the hypodermic injection of mercury is probably the quickest way of getting physiological effects, yet it has never been very popular in Great Britain. If it be employed, however, one may inject deeply into the buttock ten drops of Bloxam's solution, which contains two grains of the perchloride and one grain of chloride of ammonium to the drachm of distilled water. Chibret,[1] who has had considerable experience with the method, prefers the cyanide salt.[2] He recommends this formula:

Cyanide of mercury,	0	50 Gm.;
Hydrochlorate of cocaine,	0	25 Cm.;
Glycerin,		
Distilled water, of each,	50	0 Cc.

The average dose of this liquid is one gramme.

As a usual thing, three or four injections of either liquid, made upon consecutive days, will cause the gums to become slightly swollen and tender. If colic, tenesmus, or diarrhœa be set up, the injections must at once be discontinued.

With the advent of mild constitutional effects, the foregoing may be replaced by the simpler method of giving mercury by the mouth. It would be beside our present purpose to enumerate all the different preparations that may be exhibited, and it will be enough to state that one grain of blue pill, or a similar quantity of hydrargyrum cum creta or of calomel, administered three or four times a day, will effect everything that is either desirable or necessary. Ricord was fond of the green iodide of mercury, which is best administered in the form of a pill containing one-third to one-half grain of the salt. If diarrhœa be set up, then one grain of

[1] Transactions of the Eighth Ophthalmological Congress, 1894.

[2] A solution of the sozoiodol of mercury in iodide of potassium has been recommended by Schwimmer. Cotterell has recently praised the following combination: Sozoiodol of mercury, 5 grains; iodide of sodium, 10 grains; distilled water, 200 minims: 10–15 minims for an injection. (Syphilis: its treatment by intra-muscular injections of soluble mercurial salts, 1893.)

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Dover's powder or one-quarter grain of powdered opium should be added to each dose of the above-mentioned remedies. The fact should perhaps be mentioned that the perchloride salt is used extensively by many practitioners, and it certainly possesses some advantages when a lengthened course is necessary, as in sympathetic disease. One-sixteenth to one-sixth of a grain is often combined with two to five grains of potassium iodide in a suitable amount of water, and given two, three, or four times a day.

It is impossible to leave this subject without reminding readers of certain precautions that should be adopted in every case where mercury is about to be administered. For example, the mucous membrane of the mouth should be brought into as healthy a state as possible, while defective teeth ought to be either stopped or removed. During the actual course the patient should be warmly clad, not exposed to cold, and be careful to avoid indigestible food. He should make a point of cleansing the mouth twice or thrice daily with a solution of potassium chlorate or alum containing twenty grains to the ounce of water, and the proper and systematic use of a soft tooth-brush should be insisted upon by the surgeon. Lastly, where speedy mercurial action is imperative, the patient should be confined to bed.

Of late years the subconjunctival injection of soluble mercurial preparations has been tried upon a large scale, more especially in France and Russia. Darier, who introduced the method, asserts that excellent results follow its employment in chronic syphilitic affections of the iris, ciliary body, and choroid; it has been used, moreover, in sympathetic ophthalmitis. Either the perchloride or the cyanide salt is selected, and one to three minims of a one per cent. solution are thrown beneath the conjunctiva by means of a small syringe provided with a special needle made of so-called "iridized platinum." This little operation, which is best performed under cocaine, is followed, as a rule, by slight pain, as well as by chemosis, œdema of the upper lid, and ecchymoses at the site of puncture. It is repeated three or four days later,—that is to say, when the irritation caused by the first injection has subsided. Darier's plan is yet upon its trial, so that it is impossible to say at this moment whether it will be justified by its results. For our own part, we have reached no definite conclusion as to its value.

Some surgeons have endeavored to combat suppurative diseases of the uveal tract and sympathetic ophthalmitis by injecting various antiseptic substances into the eyeball itself. Abadie recommended that one drop or more of a one per cent. corrosive sublimate solution be thrown into the vitreous humor, while Berry employed chlorine water, Schoeler carbolic acid, and Pflüger a solution of trichloride of iron for a similar purpose.

Potassium iodide, although of comparatively little use in the acute iritis of secondary syphilis, is of distinct service in those forms of irido-cyclitis now and then met with at a later stage. At the outset five-grain doses may be given three times daily, but before long the amount may be doubled or trebled with advantage. In point of fact, it is necessary to push

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the drug freely in most cases. In order to lessen the chance of iodism, the iodide is usually combined with aromatic spirit of ammonia, and the mixture is recommended to be given in free dilution half an hour or so before a meal.

If inflammation of the iris or ciliary body be associated with rheumatism, it will, as a rule, be benefited by measures calculated to relieve that ailment. Whilst we have no intention of discussing the general management of rheumatic patients, we may briefly enumerate a few of the drugs that act well in cases of the kind. One of the most serviceable is salicine, of which fifteen to forty grains may be administered thrice a day, the liquid extract of liquorice being added to disguise its bitter taste. Salicylate of sodium, too, may be given in similar doses. The salicylate of cinchonidine (five grains, at frequent intervals) has been praised by Risley. Salol, again, is another remedy that may be tried, but, as it is insoluble in water, it is usually suspended in milk, and ten to thirty grains may be exhibited twice or thrice a day. Alkalies, particularly bicarbonate of potassium, are useful in some cases. Lastly, there is iodide of potassium, which is specially valuable in chronic irido-cyclitis. It often succeeds where the newer agents fail.

In gonorrhœal iritis the urethra must receive attention. It would lie outside our province to detail the methods of doing this, and we must content ourselves with pointing out that iodide of potassium not infrequently affords considerable relief in this type of disease. The dose should be liberal. An excellent prescription combines ten grains of the iodide with one drachm of the compound tincture of cinchona and three drachms of water, this mixture being given twice or thrice a day. Change of climate or a sea-voyage would be likely to do good in cases of the kind.

In addition to local treatment, gouty iritis calls for the adoption of those measures that have been found useful in podagra. Thus, the diet must be regulated, stimulants withheld or given with caution, and colchicum, the alkaline salts, or piperazine administered. Mineral waters are serviceable, and a visit to Buxton, Bath, Strathpeffer, Moffat, Homburg, Vichy, Royat, Wiesbaden, Carlsbad, or Aix-la-Chapelle is to be recommended.

General treatment must be also enforced in diabetic iritis or cyclitis. Salicylate of sodium has been recommended in this form of inflammation.

Malarial cases are often benefited by large doses of quinine. Arsenic, too, appears to be useful; it is best used in the form of Fowler's solution (liquor arsenicalis, B.P.), of which two to five minims may be given two, three, or four times a day.

Tubercular cases should be treated by measures calculated to improve the general health. It is almost needless to say that a liberal dietary, fresh air, and good surroundings are of the first importance. Cod-liver oil is specially indicated, and so are such drugs as quinine, arsenic, and iron. The internal or hypodermic administration of creosote or guaiacol has been

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recommended. Quint has reported two cases of tubercular iritis cured by the former remedy. Iodoform (thirty to forty centigrammes a day) has been given by Panas and others, but it should not be forgotten that this agent may sometimes give rise to retrobulbar neuritis, as in cases reported by Hirschberg, Priestley Smith, etc. If the disease continues to advance despite general treatment, the eye should be removed, although that should be seldom done unless other organs seem to be free from tubercular mischief. Still, the surgeon may be driven to enucleation, even when the disease is not primary, by great pain, by sympathetic neurosis of the other eye, or by impending perforation of the globe. Iridectomy ought never to be performed, inasmuch as fresh growths are almost sure to make their appearance.

Tonics are indicated not only during convalescence from acute iritis, but also in long-standing cases. Quinine (or one of its various preparations) is usually selected, and its administration may be continued for an indefinite time. Cod-liver oil, although not exactly a tonic, possesses considerable value in chronic inflammations of the uveal tract, especially when associated with a debilitated state of the system. It may be often combined with iron, and it seems unnecessary to remark that its dose must be regulated entirely by the digestive powers of the patient.

Surgical Treatment.—Surgical treatment may be discussed under three heads: 1. The operations done during acute disease. 2. Those performed later, in order to remedy the bad results that sometimes follow iritis and cyclitis. 3. Those carried out with the idea of preventing relapses of inflammation.

1. Two surgical operations are occasionally practised in acute iritis or irido-cyclitis,—viz., *paracentesis* and *iridectomy*. The former was recommended by von Graefe whenever there was much diffuse cloudiness of the aqueous humor, but at the present time the tendency is to restrict it more and more to cases that present exceptional difficulties in the way of cure. By some authorities, however, three conditions are recognized as justifying paracentesis. The first is when the tension of the eyeball is persistently and notably raised; the second, when a considerable hypopyon occupies the anterior chamber, or when numerous dotted deposits are present upon the back of the cornea; the last, when inflammatory symptoms obstinately refuse to yield to simpler methods of treatment. Personally, we should reserve paracentesis for the first of the foregoing classes,—that is to say, for cases in which tension remains high, despite the careful and intelligent use of myotics, such as physostigmine and pilocarpine. It matters little whether this be accompanied by hypopyon or not; the main point is to reduce the heightened tension, to which everything else must be subsidiary.

If the indications for paracentesis in acute disease are limited, those for iridectomy are still more restricted. The latter proceeding is scarcely to be thought of when there is a tendency to purulent deposits, or when numer-

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ous dotted spots are present upon Descemet's membrane, because its performance under such circumstances would probably be followed by increased inflammation and closure of the newly made pupil by lymph. But the case is different when persistent high tension occurs without great structural change in the iris, and iridectomy may then be resorted to, if paracentesis has been tried and failed.

The methods of performing paracentesis and iridectomy need not be described in this place, inasmuch as full details concerning them will be found in the article specially devoted to operations. (See present volume.)

2. Apart from the surgical management of single or multiple synechiæ (which will be considered later), operative measures will be necessary when the pupil is either "occluded" or "excluded." Nothing of the kind should be undertaken, however, until the eye has become quiescent, unless tension be raised. Iridectomy is indicated in both these cases, although for different reasons. In "occlusion" it is needed for the purpose of restoring sight, which is necessarily more or less reduced by the film present in the pupillary area; on the other hand, in "exclusion" it is called for to avert secondary glaucoma, which, as already pointed out, is the outcome of that condition when left to itself, provided the ciliary body retains its functions. The same operation is not less indicated when the two conditions coexist, but the reasons for this must be so apparent from what has been said that they need not be more explicitly stated. It is also performed when the pupil, although not actually excluded, appears to be in imminent danger of becoming so. Before proceeding to iridectomy, the surgeon must assure himself that perception of light is good. When "occlusion" only is present, he should also make a point of ascertaining whether the eye enjoyed good sight prior to the inflammatory attack, whether squint was ever present, and, lastly, whether the cornea is transparent, at least in respect of that part from behind which he purposes to remove the iris. In "occlusion" the surgical coloboma should be made preferably in the lower nasal quadrant of the iris, as the visual axis generally traverses a corresponding portion of the cornea. In "exclusion," on the contrary, it is placed, as a rule, beneath the upper lid; and so long as the communication is restored between the anterior and the posterior chamber, it is a matter of comparatively little consequence whether the cornea is clear or not.

Surgical intervention is sometimes undertaken when *total posterior synechia* is present. Iridectomy is usually practised under these circumstances, but it seldom succeeds. Three causes are responsible for this: first, the tissues of the iris are so friable that great practical difficulty is experienced in obtaining anything approaching a satisfactory coloboma; secondly, even after an apparently happy iridectomy, the uveal coat may remain attached to the lens-capsule, thus rendering null and void the best efforts of the surgeon; lastly, the lens is often more or less opaque. Hence extraction of the latter, combined with iridectomy, constitutes, upon the whole, the most satisfactory method of dealing with these difficult cases.

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At the same time, it must never be forgotten that cyclitic membranes may lie in the depths of the vitreous, so that even after a successful extraction the patient's condition may be little bettered. A cautious prognosis, therefore, should invariably be given.

The reader will perceive, then, that before proceeding to operation in any given case it is essential to make the distinction between "*exclusion*" and *total posterior synechia*, since a simple iridectomy, while benefiting the former, will almost certainly fail in the latter. The differential diagnosis between these two conditions has been alluded to more than once already, but its importance is so great from the present stand-point that it will readily bear a further reference. As indicating simple "exclusion," we have (*a*) the "crater-shaped iris," in which the central parts of the membrane are thrust forward towards the cornea, while its pupillary edge is tied down to the lens-capsule; (*b*) heightened tension of the eyeball. *Total posterior synechia* will be manifested (*a*) by marked changes in the color and texture of the iris; (*b*) by a deepening of the anterior chamber, and at times by a retraction of the root of the iris; (*c*) and, in the later stages, by a reduction of tension. Of course, in neither case does the iris respond to atropine. For reasons already given, it may, however, react slightly when exposed to light in "exclusion," although that is scarcely possible in *total posterior synechia*. This latter point, therefore, may in some instances serve to distinguish the two conditions. Finally, it is exceedingly rare to meet with total posterior synechia without "occlusion" being also present.

There are three important contraindications to the operative treatment of total posterior synechia which the surgeon will be well advised not to disregard. In the first place, when no fundus reflex can be obtained upon using the ophthalmoscopic mirror, the presumption, in the absence of visible obstruction in the pupil, is that the vitreous is blocked by cyclitic membranes: under these circumstances, operation would, of course, be futile. Again, when tension remains markedly subnormal, the eye, as a rule, is so much disorganized that surgical interference will be useless. Lastly, in order to operate with any success, a cardinal principle demands that perception and projection of light be present.

Three methods of operation lie open to the surgeon. The first differs in no essential respect from the extraction of a senile cataract, so that it need not be more particularly described in this place. The second, which is a modification of Baron de Wenzel's method of removing cataract, is, briefly, as follows. A von Graefe knife is passed into the iris through the limbus at a point about three millimetres below the transverse tangent to the upper part of the cornea. The blade is then thrust across the chamber behind the iris, and its point is made to emerge at the opposite side, after which it is pushed through the corresponding part of the limbus. By a few gentle sawing movements of the knife the section is now completed, and in this way a flap is made containing cornea, iris, and lens-capsule. The two structures last named are next seized with delicate forceps and a large

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piece of them cut away with scissors. Finally, the lens is removed either by manipulation or by aid of the scoop. If the case be complicated with "occlusion" of the pupil (as it generally is), the section should be made downward, or obliquely downward and to one side, although the latter is by no means easy to do. The third method, which was adopted by Sir William Bowman in dealing with these cases, may be thus explained. After a speculum has been inserted and the eyeball steadied by fixation forceps, a large triangular keratome is passed through the upper margin of the cornea and made to enter the substance of the iris at a point slightly beyond the pupil, so producing a transverse incision about three millimetres in length. The blades of a pair of sharp-pointed scissors are then passed into the chamber, one behind, the other in front of the iris. They are pushed downward until they reach the inner end of the transverse incision, when they are closed,—a proceeding that is next repeated from the other side. The result of this is that an oblong piece of iris and lens-capsule is included between the lateral cuts and the transverse incision. The next step is to seize this flap with iris forceps, dividing at the same time its peripheral attachments to the ciliary body. Lastly, the lens, as in Wenzel's operation, is removed.

3. A. von Graefe maintained, in his well-known memoir,[1] "that the principal cause of the recurrence of iritis was the existence of synechiæ, especially when broad and inextensible." He based this inference upon the following observations and experiments. To begin with, he found that cases not associated with adhesions seldom evinced any tendency to recur; those with slight ones relapsed comparatively seldom; while cases with many broad, unyielding synechiæ generally recurred. Then, in bilateral iritis (not syphilitic) he made the experiment of treating one eye with and the other without mydriatics. Occasionally both got well without residual synechiæ, and in that event no special tendency to relapse was observed in either. At other times, however, the eye treated without mydriatics recovered with posterior synechiæ, and recurrences were frequently noticed in it, none taking place on the other side. Graefe, without committing himself to an opinion one way or the other, suggested that adhesions might predispose to recurrent inflammations either by obstructing the local circulation or by dragging upon the iris so that it could respond but imperfectly to its natural stimuli.

It thus came to pass that various surgical proceedings were devised to prevent the evil consequences assigned to posterior synechiæ. Graefe himself practised iridectomy, but some others, among whom we may mention Streatfeild, Weber, Ogston, Passavant, and Lang, preferred to detach the adhesions by special methods of operation, to which the general name *corelysis* has been applied.

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[1] Memoirs on Iridectomy in Certain Forms of Iritis, Choroiditis, and Glaucoma, Sydenham Society's Translation, London, 1859.

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The experiences of many other surgeons, however, have not agreed with those of von Graefe; so there has latterly been a wide-spread tendency to look upon relapsing iritis as due not so much to the local condition of the eye as to diathetic causes. That this view is in a majority of the cases a just one can be scarcely doubted, for it has been shown beyond dispute that recurrences are comparatively rare in the ordinary form of syphilitic iritis, whether adhesions be left or not, and, further, that they are frequent in rheumatic cases, altogether apart from the existence of synechiæ.[1] Moreover, clinical experience has shown that while iridectomy is now and then successful in preventing relapses of inflammation, yet it fails to do so in a majority of instances. The former proves little, seeing that the tendency to relapse follows no general rule as to duration: so that it is impossible to deny that the cases in question might have remained free from recurrence even although the operation had never been performed.

From what has been said the reader will gather, then, that iridectomy is likely to be beneficial in but few cases. No surgeon, for instance, would nowadays dream of meddling with two or three adhesions, because the chances are that if left alone they would do not the least harm. He would limit his interference to instances where they were many, broad, and unyielding, and where they became more numerous with each access of inflammation. He would certainly not operate until every other means of treatment, both local and constitutional, had been tried in vain. In addition to these self-evident rules, there are three conditions the existence of which should be regarded as placing iridectomy almost out of the question. First and foremost is the presence of dotted deposits upon the posterior surface of the cornea, the so-called *keratitis punctata*. The second (hardly less important) is constituted by any marked alterations in the tissue of the iris, such as great thickening. The third is the existence of changes in the vitreous humor, as evidenced by fixed or floating opacities. These indicate that one has to do, not with a pure iritis, but with irido-cyclitis; and so it seems probable that operation will succeed according as inflammation is limited to the iris, whereas it will fail when the reverse is the case. To the foregoing Mr. Nettleship[2] has added some further contraindications,—viz., a myopic formation of the eye, and a tendency to spontaneous bleeding and to hypopyon.

However, if iridectomy be undertaken, a fair-sized piece of iris should be removed, and, other things being equal, the coloboma should be placed beneath the upper lid. It seems almost unnecessary to add that the operation should be done when the eye is quiescent.

Summary of Treatment.—The general principles of treatment, both medical and surgical, having been now passed successively in review, this

[1] In this respect anterior synechiæ have a very different significance from posterior ones. The consideration of the former, however, will fall mainly within the category of "Injuries of the Eye," and hence need not now detain us.

[2] Transactions of the Ophthalmological Society of the United Kingdom, viii. p. 94.

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article may be concluded with a summary of the means to be adopted in dealing with the main types of iritis and cyclitis.

1. *Syphilitic Cases.*—Atropine, unless contraindicated; mercury; iodide of potassium; opiates; leeches; protection of eye by bandage.

2. *Rheumatic Cases.*—Atropine; hot applications, wet or dry; leeches; iodide of potassium; salicylic acid or salicylate of sodium; salol; alkalies; quinine; Turkish or other baths; opiates; change of climate; protective goggles; eye, as a rule, not to be bandaged.

3. *Gonorrhœal Cases.*—Atropine, unless contraindicated; attention to condition of urethra; copaiba or sandal wood oil; iodide of potassium; quinine; salicylate of sodium; opiates; Turkish baths; sea-voyage or change of climate.

4. *Gouty Cases.*—Atropine, unless contraindicated; colchicum; lithia; piperazine; alkalies.

5. *Diabetic Cases.*—Atropine, unless contraindicated; treatment directed to the general state.

6. *Malarial Cases.*—Atropine, unless contraindicated; large doses of quinine; arsenic; opiates.

7. *Scrofulous and Tubercular Cases.*—Attention to general health; quinine; cod-liver oil; hypophosphites; creasote; iodoform; sea-voyage or change of climate; operative measures in certain cases,—for example, when the disease appears to be primary, or when perforation seems to be imminent.

8. *Traumatic Cases.*—Removal of foreign bodies, such as broken-down lens-matter, eyelashes, tumors, or metallic particles; atropine, only if indicated; if tension be raised, paracentesis or iridectomy; cold applications; leeches; opiates; protection of eye by bandage.

9. *Post-Febrile and Cachectic Cases.*—Atropine, if not contraindicated; tonics, as quinine, iron, strychnine, alcohol.

10. *Sympathetic Ophthalmitis.*—Atropine generally contraindicated; prolonged rest of eyes and exclusion of light; mercury (perchloride salt preferably); opium; removal of exciting eye.

DEGENERATIONS OF THE IRIS.

After long-continued inflammation the iris may become pigmented at one or several spots, a state of things for which Klemmer devised the somewhat uncouth name *iridoncosis*. (Fig. 2, Plate I.) This is generally associated with more or less thinning, so that the iris has a grayish, washed-out look. It may, indeed, be so attenuated as to permit light to pass through it at one or more places. This is especially noticeable after the resorption of syphilitic or tubercular growths. As a further result, small gaps may be sometimes seen in the substance of the iris, particularly when the pupil has become occluded by intra-uterine iritis.

Degenerative changes also commonly follow glaucoma, especially when the eye has been blinded by the heightened tension. In cases of this kind the uveal pigment appears to undergo hyperplastic changes and to creep

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round the margin of the pupil on to the surface of the atrophic iris. A similar sort of condition [1] has been described by Nettleship [2] as occurring in blind or semi-blind eyes. A case of this kind is depicted in the illustration (Fig. 1, Plate I.), which shows that a large part of the front of the iris is covered by reddish-brown pigment, the greater circumference of which is toothed in a curious and irregular way. In an eye examined microscopically by Nettleship, the appearances were found to be due, not to an exposure of the uveal coat by thinning of the iris, but to an actual extension of that layer around the pupillary margin; the pigment was covered by a thin sheet of tissue containing scattered nuclei; the iris was atrophic wherever new pigment was present.

In a case of absolute glaucoma, reported by Eales and Sinclair,[3] two dark brown globular masses were noticed in the upper and outer part of the pupillary area. The growths oscillated with every movement of the eye, while at the same time their surface showed "a fine jelly-like trembling." They were thought to be cysts the walls of which were pressed by the pigment layer of the uvea. Pathological examination confirmed the diagnosis, and further showed that the iris was atrophied, and that pigment changes such as those described above were present.

Lastly, calcareous or osseous deposits have been very occasionally found in the degenerated tissue of the iris.

From what has been said it is clear, therefore, that degeneration of the iris is a purely secondary process following chronic disease either of that tissue or of other parts of the eye.

TUMORS AND PARASITES OF THE IRIS AND THE CILIARY BODY.

A curious congenital condition has been described by more than one writer under the name of *ectropion of the uvea*. In these cases the uveal pigment seems to have overstepped its normal limits, and, instead of braiding the inner edge of the pupil merely, projects nipplewise into the anterior chamber. The usual condition is for one to ten chocolate-brown nodules to be noticed. They take their origin from the uveal zone, and range in size from somewhat less than one-fourth of a millimetre to three millimetres. Their seat of election is at the upper or the lower margin of the pupil. They do not hamper the movements of the iris. In rare instances the pigment becomes detached, so that particles of it lie free in

[1] Judging from the following extract, such cases did not escape the notice of the earlier observers: "In a case of acute syphilitic iritis, with a large effusion of lymph, the part of the iris which had been covered by the lymph was left, after its removal, of a dark black color. It was a little elevated above the level of the iris, and of triangular figure, with the basis at the pupil and the apex at the edge of the cornea."—W. Lawrence, A Treatise on the Diseases of the Eye, London, 3d edition, 1844, p. 403.

[2] Transactions of the Ophthalmological Society of the United Kingdom, vols. v. and vi.

[3] Transactions of the Ophthalmological Society of the United Kingdom, vol. xvi., 1896, p. 56.

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the anterior chamber (Bock); even cysts may be formed by the degeneration of such particles (Businelli, Fuchs). Although ectropion of the uvea is not uncommon, yet the projections, by reason of their small size, must often escape notice. It is natural to the eye of the horse and allied animals, and when occurring in man must be regarded as a reversion in type to the anatomical structure of vertebrates lower in the scale of creation.

An allied condition goes by the name of *melanoma*. It is due to the proliferation of the iris-stroma as distinguished from that of the uvea. These small, dark growths are by no means common, but they possess considerable interest, inasmuch as they have been shown to become in certain instances the starting-points of melanotic sarcomata. As a rule, however, they are perfectly benign. They appear to be congenital.

The nodular growths of *syphilis*, of *tubercle*, and of *ophthalmia nodosa* have been described in an earlier part of this article.

The tubercles of *leprosy* have been seen upon the iris, in which they set up inflammatory reaction. The condition is seldom primary, and follows, as a rule, corneal leprosy. The prognosis is unconditionally bad.

Cystic tumors of the iris are divided into two varieties,—namely, the serous and the epidermoid. The former have limpid contents, whereas the latter are filled with atheromatous material. Both forms are lined by epithelium, and their walls are composed of thinned and altered iris-tissue.

Serous cysts generally originate in the sinus of the anterior chamber, where they appear under the form of a small, transparent vesicle. They are usually single and limited to one eye. They slowly increase in size, and become lobulated and flattened against the posterior surface of the cornea, which they render locally opaque. A few attenuated iris-fibres not infrequently cross the front of the cyst, the anterior wall of which is often thin enough to allow one to recognize its posterior (or uveal) boundary. Grayish-brown particles of pigment flecking the wall of the tumor are sometimes observed. (Plate I., Fig. 3.) In the course of their further development, cysts give rise to distortion of the pupil, which becomes oval, kidney-shaped, irregularly triangular, or slit-like. The iris is now and then retroverted. Irido-dialysis has been described more than once; indeed, the crystalline lens may be displaced by the growing cyst, and may at a later stage become opaque. The growth may obstruct the pupillary area completely, thus reducing sight to mere perception of light. If not interfered with, these cysts ultimately give rise to glaucoma. Instances have been recorded, moreover, in which iritis or irido-cyclitis was observed, while sympathetic ophthalmitis of the other eye has been noted in a few cases (Tyrrell, Hulke, etc.).

The epidermoid cyst resembles a small seed-pearl in appearance, starts from almost any part of the iris, and grows very slowly. In the long run, however, it produces a train of secondary symptoms similar to those described above.

Both serous and epidermoid cysts are exceedingly rare. At least three-

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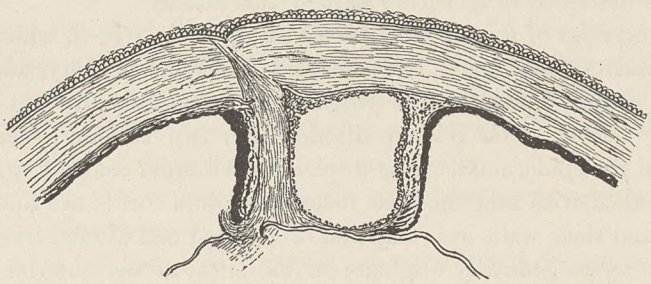
fourths of the cases develop some months or years after a penetrating injury of the eyeball, as evidenced both by the history and by the discovery of a cicatrix, which generally lies at or about the sclero-corneal margin. In the remaining cases no evidence is forthcoming of any previous injury, although that obviously does not exclude the possibility of such having taken place. It was suggested by Rothmund that particles of epithelium, derived from the cornea, the conjunctiva, or the skin, were carried into the substance of the iris upon the point of the penetrating instrument, and that they there proliferated and thus became the starting-point of cystic formation. This hypothesis has been borne out both by experimental and by clinical evidence. Cilia have been purposely implanted into the anterior chamber of rabbits, with the result that epidermoid or pearl-like tumors

FIG. 8.



So-called cyst of the iris, formed after an injury to the cornea. (Alt.)

have subsequently developed, while a similar result has followed the introduction of morsels of living conjunctival or corneal tissue (Masse). Again, there are many cases recorded where lashes have been carried into the eye by some accidental injury and in the course of time growths have developed from the cells of the root-sheath. On the other hand, McGillivray[1] has published an instance where no tumor followed the implantation of an eyelash that had lost its bulb, notwithstanding the fact that it had lain in contact with the anterior surface of the iris for eighteen months.

An attempt has been made to account for some of these traumatic cysts by assuming that they result from the sacculations brought about by synechiæ. Posterior synechiæ, so it is supposed, cut off a limited portion of the posterior chamber, the little bag becoming converted into a regular cyst by the gradual secretion of fluid into its interior (de Wecker). A similar kind of result comes about in anterior synechiæ by a fold of iris adhering to the hinder surface of the cornea. Fig. 8 represents a case in which this took place after injury to the eye. As can be perceived, the walls of the cyst are formed in front by Descemet's membrane, behind and laterally by the altered and adherent iris. The growth, it may be added, was lined by the endothelium of the anterior chamber. It has been conjectured that at a later stage cysts formed in this way may free themselves from their attach-

[1] Transactions of the Eighth International Ophthalmological Congress, 1894, p. 284.

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ments to the cornea; but it seems unlikely that this is the etiology of those cysts which are now and then seen free in the anterior chamber. It is more probable that some of these are cysticerci, and that others, as noted previously, are due to degenerative changes occurring in particles of pigment before or after detachment from the uveal layer of the iris.

Cystic formations have been noted after operations such as cataract extraction and iridectomy. In a singular case reported by Wordsworth,[1] a large number of small, globular, pearl-like bodies were discovered some three years after the discission of a lamellar cataract. Some were attached to the edge of the iris, forming a more or less complete fringe, while others were connected with an opaque membrane in the lower third of the pupillary space; a third set were lying free in the anterior chamber, looking as though they had become detached from the iris. Although most of the recorded cases have been met with in children, yet cysts have been also known to follow removal of senile cataract. The growths seem to arise from particles of epithelium carried into the anterior chamber by the instruments of the surgeon.

A few cysts appear to be of congenital origin, and it is possible that some of these may be of dermoid nature. A case of this kind was recorded by Graefe, who discovered a number of short hairs among the pap-like contents of the growth. Possibly, too, White Cooper's[2] case, in which the cyst "was as tough as cartilage," belonged to the same category.

Schmidt-Rimpler has suggested that some of the non-traumatic cysts may result from closure of the crypts normally present upon the surface of the iris. An accumulation of fluid is then assumed to take place, so that a retention—or more correctly, perhaps, an exudation—cyst is formed. It has also been surmised that some of the congenital cases may be accounted for by fluid that has collected between Descemet's membrane and a pupillary membrane (Giraud-Teulon). Lastly, Berry[3] believes that the serous cyst is "a kind of cystoid degeneration of the iris, leading to the formation of a diverticulum at the angle of the chamber."

Instances have been known in which a serous cyst has not recurred after a simple puncture, but it would, of course, be useless to attempt that operation in the epidermoid variety. Eversbusch[4] practised the following method in a case that presented unusual difficulties. An incision seven millimetres in length was made through the cornea at a point opposite to the growth, the salient part of which was at the same time punctured. Introducing Wecker's scissors through the corneal wound, he next passed the sharp-pointed blade in front of and the other blade behind the iris. By closing the instrument the cyst was divided radially, so that its two halves fell apart. This separation was kept up for some days by the employment

[1] Transactions of the Ophthalmological Society of the United Kingdom, vol. i. p. 23.
[2] On Wounds and Injuries of the Eye, London, 1859, p. 189.
[3] Diseases of the Eye, 2d edition, 1893, p. 266.
[4] Klinische Monatsblätter für Augenheilkunde, August, 1893.

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of a mydriatic. As a rule, however, a simpler method of treatment is adopted: the anterior chamber is opened with a von Graefe's knife, the tumor is seized with delicate forceps and drawn out of the wound, and the piece of iris to which it is attached is snipped through by means of curved scissors. This operation should be done under full antiseptic precautions, since it has been sometimes followed by panophthalmitis, as in a case mentioned by Graefe.[1]

A few instances of *vascular tumors* affecting the iris have been published. Mooren's is perhaps the best known case; it is certainly one of the most remarkable. A tumor was attached to the outer part of the right iris and touched the posterior surface of the cornea. It resembled a blackberry, both in size and appearance. When the patient's head was shaken and bent forward, the whole of the anterior chamber rapidly filled with blood, presumably derived from the dilated vessels of the tumor. The extravasation disappeared in the course of one and a half minutes at the outside. After dwindling to one-third of its former size, the neoplasm set up glaucoma, for the relief of which iridectomy was performed; but the second eye ultimately became affected by sympathetic disease. It is singularly unfortunate that the growth was not submitted to microscopic examination. Schirmer[2] observed a small tumor of the iris that had a cavernous structure. It was of gelatinous appearance, obviously traversed by vessels, and speckled over with red, hemorrhagic spots. Wolfe[3] has recorded a case in some respects like that of Mooren. He found upon the inner side of the right iris of a man sixty-two years of age a lobulated growth of a dark raspberry color. The tumor extended from the pupillary to the ciliary zone, encroaching upon Descemet's membrane, to which it appeared to be attached. At intervals of from four to six weeks it bled so freely as to fill the anterior chamber with blood, which became absorbed in a week or ten days. The growth was not removed. Lastly, Berry has met with a nævus which involved not only the iris but also a persistent pupillary membrane that happened to be present.

Although primary *sarcoma* of the iris is certainly a rare affection, a good many cases have been placed on record. Both pigmented and non-pigmented growths have been described, but the former have been met with more frequently than the latter. Females appear to be more subject to the disease than males. A majority of the published cases have occurred between the ages of thirteen and forty years. But Romiée[4] saw an instance in a female seventy-four years of age. The condition is generally unilateral. Carter,[5] however, brought a case under the notice of the Clinical Society in which both irides were affected with round-celled sarcomata: one tumor

[1] Archiv für Ophthalmologie, xii. 2, S. 230.
[2] Handbuch der gesammten Augenheilkunde, Bd. iv. S. 551.
[3] Medical Times and Gazette, May 8, 1880.
[4] Recueil d'Ophtalmologie, Avril, 1881.
[5] Transactions of the Clinical Society of London, vol. vii.

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was present upon the left and two upon the right iris. As noted before, sarcomata sometimes originate from a congenital pigment patch in the iris, the so-called melanoma.

Both leuco- and melano-sarcomata commence, as a rule, in the lower half of the iris. They are not transparent, but have a yellowish or brownish appearance. The non-pigmented growths often show a large number of small vessels coursing over and through them. Repeated hemorrhages have been noted from both kinds of sarcoma. This was so in a case related by Wardrop[1] as long ago as 1818. That author spoke of a small pigmented tumor growing from the iris and being so extremely vascular that "frequently, without any external cause, it bled profusely, and would in a short time fill the anterior chamber with blood." As in a couple of instances reported by Little,[2] the bleeding may give rise to temporary blindness.

Both forms of growth may remain confined to the iris for a considerable length of time, even for a long term of years; but sooner or later they take on more rapid development, setting up iritis, and giving rise to considerable irritation of the eye and disturbance of sight. Eventually they cause increased tension, perforate the globe, and diffuse themselves among the surrounding structures. Metastatic growths have been found in distant organs of the body, such as the liver. Sarcomata of the iris, when not removed by operation, cause death.

The growths have been treated successfully by performing iridectomy, thereby removing both the tumor and the portion of iris to which it was attached (Arlt, Kipp, Knapp, Little, Kruckow). This operation, therefore, may be attempted so long as the sarcoma is small and strictly limited to the iris; but if there be any doubt as to these points, the better plan will be to enucleate the eye without further loss of time.

The ciliary body may be the primary seat of a new growth; it may, upon the other hand, be affected secondarily to some other part of the uveal tract. It may also be involved metastatically, as in a case recorded by Ewing,[3] where carcinoma of the breast was followed by a secondary growth of the iris and the ciliary body. Excluding tubercle and gummata, primary tumors of the ciliary body are exceedingly rare, as will be gathered from the statement that literature contains only about thirty-five cases of the kind.

A primary tumor of the ciliary body sooner or later causes iridodialysis, and makes its appearance in the anterior chamber, where it gives rise to repeated hemorrhages. Before this takes place, however, it causes the patient little uneasiness, although should the case come under notice its existence may be inferred with more or less approach to certainty. Thus, by focal illumination the surgeon may see the neoplasm as a darkish mass

[1] Essays on the Morbid Anatomy of the Human Eye, vol. ii. p. 49.
[2] Transactions of the Ophthalmological Society of the United Kingdom, vol. iii.
[3] Archiv für Augenheilkunde, 1890, S. 121.

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lying behind the iris, or during the course of ophthalmoscopic examination he may observe a growth springing from the ciliary region of the fundus. The diagnosis will be by no means so simple if the surgeon has to do with a painful eye, with raised tension, but no actual evidence of tumor. In some of these cases the retina may be found detached, or it may be impossible to elicit a reflex from a particular area of the ciliary zone. Under these circumstances an intra-ocular growth is probably present, a diagnosis that will be strengthened if the eye be also blind. There will be small room for doubt as to the nature of the case if, along with the foregoing signs, "scleral" or "episcleral" nodules are present. These brown or blackish spots (which must not be confounded with congenital pigment changes) are found in the neighborhood of the limbus, around the points where the anterior ciliary vessels enter or leave the eye. They indicate that the cellular elements of the neoplasm have penetrated the globe and proliferated externally.

Ciliary tumors, like all intra-ocular growths, eventually give rise to heightened tension. This is succeeded by perforation of the eyeball in the ciliary region of the sclera, an event naturally associated with lowered tension. If the tumor be malignant—which, practically speaking, is always the case—this is speedily followed by infection of the neighboring structures, as, for example, the eyelids and the tissues of the orbit. Apart from this, distant organs may become affected.

The following tumors have been described as originating from the ciliary body:

1. Sarcomata. Round, spindle, mixed, myo-, myxo-, and cystic sarcomata have been met with. In a majority of the specimens submitted to microscopical examination more or less pigment was found in or around the cells of the growth.

2. Myoma and myo-fibroma. See the cases published by Iwanoff,[1] Solomon,[2] and Felix Lagrange.[3]

3. Carcinoma (Badal and Lagrange,[4] E. Treacher Collins[5]).

4. Angioma (A. H. Griffith[6]).

The only method of treatment consists, of course, in removing the eyeball as soon as the diagnosis is satisfactorily established.

Parasites.—The measle of taenia solium has been found both in the anterior chamber and upon the iris of man. Mackenzie[7] quoted five cases of the kind, and since he wrote a good many others have been published. In typical instances a small, semi-transparent, spherical body lies at the

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[1] Described by Wecker in the Handbuch der gesammten Augenheilkunde.

[2] Transactions of the Ophthalmological Society of the United Kingdom, vol. ii. p. 263.

[3] Études sur les tumeurs de l'œil, Paris, 1893, p. 81.

[4] Archives d'Ophtalmologie, 1892, p. 143.

[5] Transactions of the Ophthalmological Society of the United Kingdom, vol. xiv.

[6] Medical Chronicle, April and May, 1892.

[7] Treatise on Diseases of the Eye, 4th edition.

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bottom of the anterior chamber or floats in the aqueous humor. At one particular spot a whitish projection is noticed, which changes its shape from time to time either spontaneously or under the influence of movements of the patient's head. Iritis is set up sooner or later. If not removed, the cysticercus eventually entails loss of the eye.

The filaria sanguinis (? Bancrofti) also has been met with in the anterior chamber. Barkam[1] removed such a parasite from the iris of a man, thirty years of age, who had lived in Australia. Macnamara[2] and Drake-Brockman[3] have seen cases of the kind in India. In a remarkable instance recently described by H. Coppez,[4] one of these worms—one-half millimetre thick and three centimetres long—was observed moving about actively in the limpid aqueous humor of an infant lately brought from the Congo. The animal died, became coiled at the bottom of the anterior chamber, and was extracted through a corneal incision. The specimen was examined by Van Duyse. Gauthier[5] has related the case of a young negro in whose anterior chamber a filament two centimetres in length and one millimetre in thickness was seen revolving with great rapidity. According to his account, the filament was ordinarily folded in half, and manifested a rapid undulation through its entire length.

DISORDERS OF MOVEMENT OF THE IRIS.

Although it would be out of place to embark upon anything like a physiological discussion, we may nevertheless point out that considerable divergence of opinion exists with regard to the muscular mechanism of the iris. It has been found experimentally that the pupil becomes smaller when the oculo-motorius is irritated or the sympathetic is divided, while it becomes larger when the oculo-motorius is divided or the sympathetic is irritated. Hence the widely held view that the movements of the iris are controlled by two muscles, namely, the sphincter and the dilator pupillæ, the former being innervated by the oculo-motor and the latter by the sympathetic. Now, in man the sphincter can be readily demonstrated. It forms a narrow ring of smooth muscular fibres encircling the pupil and lying close to the posterior surface of the iris. There is, then, no dispute upon this point; but with regard to the so-called dilator the case is different. Such a muscle undoubtedly exists in certain animals, as birds and otters, and a few observers assert that it may be found in the human eye, where, according to them, it forms a flattened layer of smooth muscular tissue extending from the attachment of the iris nearly to the pupil, close to the posterior surface of the former (A. E. Schafer[6]). Others, while admit-

[1] Archiv für Augenheilkunde, 1876, S. 381.
[2] Indian Annals of Medical Science, vol. viii. p. 405.
[3] Medical Press and Circular, October 24, 1894.
[4] Archives d'Ophtalmologie, xiv. 9, p. 557.
[5] Annales de l'Institut de Chirurgie de Bruxelles, 1895.
[6] Essentials of Histology, London, 1892, p. 266.

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ting the existence of such a layer, deny its muscular character, and hold that it is composed of elastic fibres merely (Fuchs). Another school denies that it exists in man. Langley and Anderson,[1] who have recently investigated the matter, conclude as the result of their experiments that dilatation of the pupil is due to the contraction of some radially disposed substance in the iris, although they are not prepared to state precisely what that substance is. Finally, there is a good deal to be said in favor of Gaskell's theory,[2] which assumes the sphincter pupillæ to be the only muscle present in the human iris. Normally it is supposed to be in a state of tonic contraction, which is increased by the catabolic influence of the oculo-motor (contraction of the pupil), and diminished by the anabolic influence of the sympathetic (dilatation of the pupil). This inhibitory hypothesis, as it may be termed, seems to be supported by the fact observed by Grünhagen, that direct electrical excitation of the excised iris might give rise either to lengthening or to shortening. As it does not clash with clinical experience, it forms a good working theory.

Disturbances of movement may be discussed under five heads: (1) *mydriasis*, (2) *myosis*, (3) *hippus*, (4) *iridodonesis*, and (5) *alterations in the muscularity of the iris.*

MYDRIASIS.

Mydriasis, or abnormal dilatation of the pupil, may be either active or passive; that is to say, it may result from irritation of the sympathetic or from paresis or paralysis of the oculo-motor nerve.

Active mydriasis is not common. The dilated pupil reacts both to light and to accommodation. It has been described as forming a symptom of certain nervous diseases, such as cerebral or spinal meningitis and intracranial tumors. It is present also when the cervical sympathetic is irritated by wounds, new growths, or in other ways. It is said to be met with as a reflex sign in some disorders of the intestinal and genital organs. The mydriasis produced by cocaine appears to be of this nature, while that of the anæmic and chlorotic states, of typhoid fever, and of migraine probably owns a similar cause. Active dilatation of the pupil, as everybody knows, accompanies anger, fear, and some other emotions of the kind. Lastly, there is an interesting dilatation, apparently due to psychical influences, which is sometimes seen in children or young persons of nervous temperament. The mydriasis, seldom complete, usually affects one eye, and may come on while the patient is under observation. As a rule, it speedily subsides, but it may persist for weeks. It is exceedingly apt to recur. It is now and then associated with cycloplegia, in which case the pupil is widely dilated and has lost its direct action to light.

Passive mydriasis falls more commonly under the notice of the surgeon than the form just described. Since it is the result of paresis or paralysis

[1] Journal of Physiology, Nos. 5 and 6.
[2] Ibidem, No. 7, p. 38.

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of the oculo-motor nerve, it is often associated with cycloplegia. The following are some of the more important conditions in which passive mydriasis is found:

(1) Paralysis of the third cranial nerve. The pupillary branch may be alone affected, though usually other parts of the nerve are also involved.

(2) Some cerebral affections,—*e.g.*, progressive paralysis of the insane, tumors or other lesions in the neighborhood of the third nerve nucleus, thrombosis of the cavernous sinus.

(3) The local application or internal administration of agents like atropine, homatropine, duboisine, daturine, and scopolamine.

(4) Poisoning by the products of the diphtheria bacillus, or by the ptomaines found in decaying articles of food, such as sausages.

(5) When the ciliary nerves have been damaged by the heightened intra-ocular tension of glaucoma.

(6) Syphilis may be the cause of unilateral mydriasis.

(7) After injuries, such as blows upon the eye. The mydriasis in these cases may persist for years, and is often associated with lowered tension.[1]

MYOSIS.

Like mydriasis, myosis may be either active or passive. In other words, it may be due to irritation of the sphincter pupillæ or of its nervous supply, or to paralysis of the sympathetic.

Active myosis follows the local use of agents like physostigmine and pilocarpine, or the internal administration of morphine, nicotine, and some other drugs. It is seen in the earlier stages of meningitis and apoplexy. It has been described as existing in cases where a new growth was setting up irritation of the third cranial nerve. It has been regarded as a premonitory symptom of an impending hysterical or epileptic seizure. It is said to occur, as a sort of trade spasm, among those who employ their eyes upon minute objects for long together. As already pointed out, it forms one of the signs of hyperæmia or inflammation of the iris.

Passive myosis is met with in paralysis of the cervical sympathetic. In that case it is associated with some or all of the following signs: slight drooping of the upper lid, enophthalmos, reduced intra-ocular tension, and alterations in the vascularity of the corresponding side of the face. Cocaine, when applied to the eye, produces neither retraction of the upper lid nor dilatation of the pupil. This condition has been known to follow injuries

[1] The mydriasis associated with optic atrophy has not been included in the foregoing list, inasmuch as it has nothing to do with the third nerve. It is due, of course, to a break in the transmission of the light impulses.

Somewhat similar considerations apply to the dilatation of the pupil that may follow contusion of the eyeball. The mydriasis in these cases seems to be generally due to a small rent in the pupillary edge of the iris, which weakens the sphincter muscle to a greater or less extent. Traumatic mydriasis is usually partial, so that the pupil is unequally dilated. It is often associated with reduced tension and with paralysis of the accommodation.

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of the neck,—for example, stabs, gunshot wounds, and surgical operations; it has also resulted from the pressure of aneurismal or glandular swellings.

Myosis is frequently found in the early stages of locomotor ataxia, but in this case is probably due to degenerative changes affecting the centre for dilatation of the pupil. This so-called spinal myosis is often so exceedingly marked as to justify the term "pin-point" pupil. It is generally bilateral. The pupil at first reacts both to light and to accommodation convergence, but at a later stage it may respond to the latter alone. When this is the case the condition is spoken of as the Argyll-Robertson[1] phenomenon, in honor of its discoverer. It need scarcely be added that the pupillary signs of locomotor ataxia are generally found associated with other ataxic symptoms, as, for instance, loss of the tendon reflex, alterations in the visual field, optic atrophy, fleeting paralyses of the eye and other muscles, unsteady gait, lightning pains, deafness, constipation, and disturbances of the genital and urinary tracts.

HIPPUS.

As may be seen by the use of a magnifying glass, the healthy iris is in a state of constant oscillation, so that the pupil is continually undergoing slight changes in diameter. This physiological condition must not be confounded with a pathological hippus, in which the iris manifests an intermittent or clonic spasm sufficiently marked to be appreciated by the unaided eye. This latter state of affairs is very uncommon. It has been met with in meningitis, in multiple sclerosis, and in some other affections—both organic and functional—of the nervous system. It may coexist with nystagmus, as shown by three cases published by Jessop in the seventh volume of the "Transactions of the Ophthalmological Society." Gunn[2] states that hippus now and then marks the point at which sympathetic neurosis is about to yield to sympathetic ophthalmitis; but this observation does not appear to have been confirmed by other surgeons.

IRIDODONESIS.

A slight trembling of the iris, as already pointed out, may be occasionally noticed in perfectly normal eyes, especially when the pupil is greatly contracted. This must be distinguished from the pathological condition spoken of as *iridodonesis*. The latter can occur only when the iris has lost the support of the crystalline lens,—as, for instance, when the last-named structure is luxated, congenitally deficient, or absent. In these circumstances the whole or some particular part of the muscular curtain manifests a marked oscillatory movement when the eye is quickly turned from side to side. Its quivering has been likened to that undergone by a piece of cloth exposed to a fluctuating wind. To detect iridodonesis, the

[1] Edinburgh Medical Journal, 1869, p. 703.

[2] The Students' Guide to Disease of the Eye, Nettleship, 4th edition, 1887, p. 142 (foot-note).

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patient is directed to turn his eyes rapidly from one side to the other, and then to look at the surgeon's uplifted finger held a short distance away from his face. The position of the tremulous iris shows, as a rule, some departure from the normal. Thus, instead of bulging slightly forward, as in the healthy eye, it may hang vertically or even be drawn backward towards the vitreous chamber. It was formerly thought that synchysis of the vitreous was enough to give rise to iridodonesis. It is now generally believed, however, that the mere fluidity of that humor can have no influence whatever upon the iris, provided the lens occupies its natural position and the zonula remains intact. When the latter has undergone alterations, lens as well as iris may be tremulous.

ALTERATIONS IN THE MUSCULARITY OF THE IRIS.

The pupils of those with congenital cataract often react imperfectly to mydriatics. Altogether apart from disease, however, there appear to be great differences in the muscularity of the iris among individuals. Thus, it is not uncommon to find that the pupils, even after correct and repeated applications of atropine, remain smaller than usual. Three hundred and sixty-eight children under the age of seventeen years were recently examined for the purpose of obtaining precise figures. Of that number no fewer than seventy-seven (or twenty per cent.) were found to have pupils the transverse diameter of which was seven millimetres or less. Anisocoria was not observed. The condition appears to run in families.

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DISEASES OF THE CHORIOID AND VITREOUS.

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PART I.—DISEASES OF THE CHORIOID.

BEFORE discussing the affections of the chorioid, it will be advisable briefly to refer to some of the most important practical points in the anatomy and pathology of this structure.

The chorioid is a very vascular pigmented and elastic membrane which lines the inner surface of the sclerotic from the entrance of the optic nerve, where it arises as a ring, to a zone corresponding to the insertion of the recti muscles and the termination of the true retina at the ora serrata. It is here directly continuous with the ciliary body and the iris, forming with them the uveal tract.

It is thickest behind near the optic disk, measuring here, inclusive of the uveal pigment-layer, about one-sixtieth of an inch; and we must recollect that during life it measures probably several times as much as this.

Except around the optic disk and at the region of the macula, the chorioid is only loosely attached to the sclerotic by vessels, nerves, and delicate connective tissue, a lymph-space intervening which communicates on the one hand with the intervaginal space of the optic nerve, and on the other hand with the lymph-space subtending Tenon's capsule, which it reaches along the blood-vessels which perforate the sclerotic at the equator.

On cutting open eyes in a sagittal direction, the anterior part of the chorioid separates from the sclerotic on account of its elasticity. The rest of the chorioid shows no spontaneous tendency to become detached, though it may readily be completely separated by a little traction, the connection with the sclerotic, although very feeble, being yet a close one.

The inner surface of the sclerotic thus exposed shows a delicate pigmented connective tissue, the *lamina fusca*, which levels up all inequalities and grooves caused by the presence of vessels and nerves, and extends pretty deeply into the substance of the sclerotic, especially near the entrance of the optic nerve.

The inner surface of the chorioid, when exposed to view by removal of the retina, will always be found closely invested by the hexagonal pigment-

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layer of the retina, which for all practical purposes may be regarded as belonging to the chorioid, being dependent upon the latter for its nutrition, and showing early disorganization in all chorioidal inflammations.

In describing from within outward the structure of the chorioid we have, as seen for the most part in Fig. 1,—

(1) The *layer of hexagonal pigment-cells.* Seen in a surface view, they form a complete mosaic of six-sided pieces fitted together quite closely. In sections one often finds this layer lifted off the chorioid as an unbroken sheet for long distances, the cells being evidently very closely cemented together.

The individual cells are cylindrical in form, the end presented to the retina being rather the larger and showing a rounded apex. The pigment-granules are mostly crowded towards this end; the other extremity shows a single large rounded nucleus, and rests by its flat base on the next layer, the lamina vitrea.

This description applies only to anatomical sections, for during life and under the influence of light the cells change their form and send long processes into the outer layer of the retina; the pigment-granules also change their position in the cell, following the prolongation into the retina.

(2) The *lamina vitrea* is a structureless membrane which faces the chorioid in its entire extent. It extends well on to the disk beyond the other layers of the chorioid; its inner surface is quite smooth, sharply defined, and free; the outer surface sends processes at right angles into the inner layers of the chorioid, which unite it firmly to that structure and form loops or channels in which run the capillary vessels of the chorio-capillaris.

(3) The *chorio-capillaris.* The capillary net-work here forms a single layer of vessels, and is said to be closer than in any other part of the body.

(4) The *tunica vasculosa* consists mainly of blood-vessels united by a delicate connective tissue which contains numerous large ramified and pigmented cells. The arteries are easily distinguished in section from the veins by the thickness of their walls and the presence of a muscular coat.

Most of the pigment of the chorioid is found in this layer, especially behind and between the blood-vessels, the anterior aspect of the larger vessels being nearly free from pigment. The coloring matter is contained in flat, branching, spindle-shaped, and stellate cells which are arranged with their surfaces parallel to the chorioid. The granules are evenly distributed throughout the body of the cell, except at the nucleus, which is nearly free.

(5) The external layers of the chorioid, forming the *lamina supra-chorioidea,* are much more dense and less vascular than the foregoing; they consist of several homogeneous membranes pervaded by elastic fibres and covered by large, flat, epithelium-like cells and irregular areas of large, flattened pigmented cells.

The functional activity of the rod-and-cone layer of the retina is dependent upon the hexagonal pigment-layer, and this latter will be found

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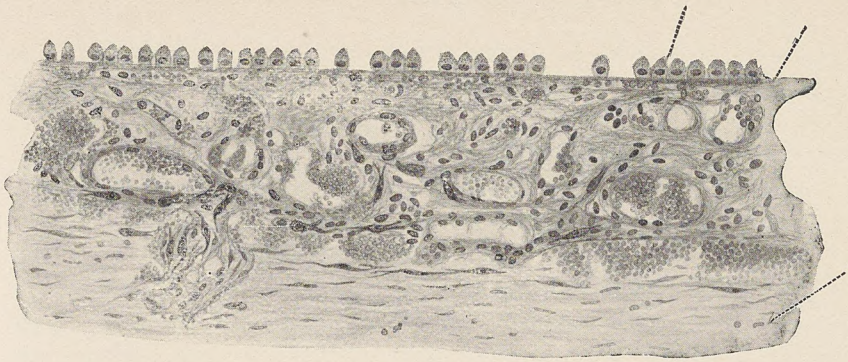
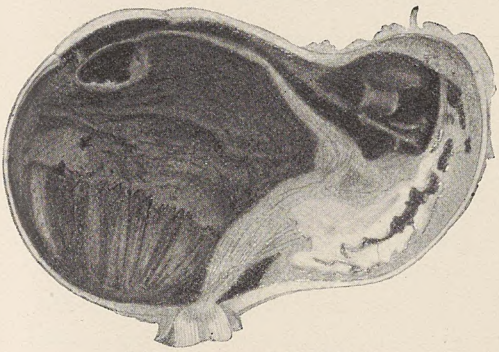
Fig. 1.



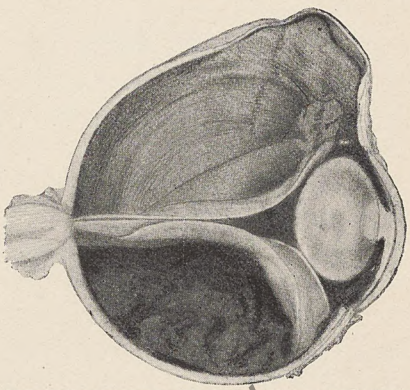
Section of normal human adult chorioid with retinal pigment layer and sclerotic.

Fig. 2.



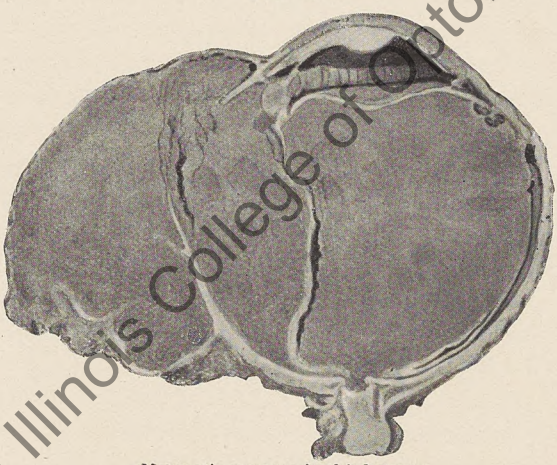
Tubercular growth in chorioid.

Fig. 3.



Melanotic sarcoma with large ciliary staphyloma away from growth.

Fig. 4.



Melanotic sarcoma in third stage.

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more densely pigmented where it corresponds to the macula lutea, so that on stripping off the retina a very dark round spot can nearly always be seen here.

The blood-supply of the chorioid is mainly from the short ciliary arteries which perforate the sclerotic near the entrance of the optic nerve; the veins lie external to the arteries, and converge to form four or five trunks which perforate the sclerotic at the equator.

The function of the chorioid is to maintain the nutrition of the outer layers of the retina, which have no blood-supply of their own; the general nutrition of the eyeball and vitreous humor, on the other hand, being directly dependent upon the ciliary body. The consequence of this is that disorganization and shrinking of the entire globe usually ensue from cyclitis, but not from uncomplicated chorioiditis.

INFLAMMATIONS OF THE CHORIOID.

Inflammation of the chorioid may be either *plastic* or *purulent* in type: the former affects the chorioid only, and is recognized by the ophthalmoscope; the latter never remains limited to the chorioid, but speedily produces purulent infiltration of the vitreous and shrinking of the globe.

Purulent Chorioiditis.—Purulent chorioiditis, when traumatic in origin, is caused by direct septic infection, as in penetrating wounds or operations on the globe, especially cataract extraction; in sloughing and perforating ulcers of the cornea, under which circumstances one eye only will be affected; it may also occur as a metastatic affection, when both eyes are often attacked, one shortly after the other; in surgical or post-partum pyæmia, especially the latter, and in septic endocarditis likewise, but usually in a less acute form; at the termination of certain acute disorders, as influenza, scarlet fever, measles, diphtheria, scorbutus, variola, etc.; in erysipelas, typhoid, pneumonia, and cerebro-spinal meningitis, epidemic or sporadic; and also, in a very subacute form, in children under two years of age, after acute but transient cerebro-spinal symptoms.

Cases have also been recorded after trivial surgical conditions, as extraction of a tooth, suppuration in the alveolar cavity of a tooth, purulent finger-joint, etc.

Symptoms.—Purulent chorioiditis or panophthalmitis is characterized by pain in the eye and head, intense inflammation of the eye, and rapid and total loss of sight. It is often ushered in by severe headache and vomiting, rise of temperature, and general febrile symptoms.

The entire conjunctiva becomes deeply injected, thickened, and inflamed, the cornea yellowish and hazy, the aqueous muddy, the iris inflamed and adherent to the lens-capsule, with the presence of pus in the anterior chamber; the globe becomes more or less protruded and limited in its movements, from the inflammation having extended to the tissues around it; the eyelids are swollen, red, and infiltrated, often forming with the globe a swelling as large as a Tangerine orange.

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The tension is usually increased, but only for a short time. At the height of the inflammation and pain the pus breaks through the sclerotic near the equator or between this and the cornea, the pain speedily abates, and the globe shrinks.

The whole process takes from four to six weeks to run its course.

The symptoms may in some cases abate without perforation, consolidation and partial absorption of the intra-ocular exudation taking place, and if the cornea remain fairly clear we may even be able to see something of the yellowish lymph through the pupil; but the sight is in all cases totally destroyed.

Pathology.—The disease is always due to septic infection; micro-organisms can nearly always be found; but even if these are absent the causation is probably the same, for, as Leber has shown, suppuration may be brought about by substances derived from the staphylococcus aureus as well as by that organism itself.

The micro-organisms found are the staphylococcus albus, aureus, and citreus, and the streptococcus pyogenes; bacterial thrombi have been demonstrated in the vessels of the chorioid, iris, and ciliary body, retina, conjunctiva, muscles, and orbital contents.

The inner layers of the chorioid near the lamina vitrea are packed with accumulations of pus-corpuscles, the other pigmented and deeper layers being very free from these; but the lamellæ are widely separated from one another, and extravasations of blood are often present.

The veins are thrombosed and the arteries plugged with emboli, and there is hyaline swelling of the smaller vessels.

There is a cloudy or finely granular exudation on the inner surface of the lamina vitrea, with the retinal pigment-cells scattered throughout in heaps.

The lamina vitrea may be broken through from exudation on its outer surface, and when this occurs the edges of the rupture curl inward.

The retina is thickened and beset with pus- and blood-corpuscles, and its vessels are plugged with bacterial emboli. The nuclear layers are little changed, but widely separated by expansion of the internuclear layer, which shows large empty spaces, possibly from accumulation of serum which has escaped.

The internal limiting membrane presents a wavy surface, and is strongly bulged towards the vitreous by the presence of fibrillar tissue and blood in the greatly thickened nerve-fibre layer. The rods and cones and ganglion-cells disappear, the retina being recognized by the two nuclear layers.

The space of Tenon is obliterated by the capsule becoming adherent to the outer surface of the sclerotic, and dense vascularized masses of inflammatory origin pervade the tissues of the orbit around the globe and are found firmly adherent to the outer surface of the sclerotic.

The vitreous is invaded by extensive areas of purulent exudation, arising from the ciliary body and the inner surface of the retina, chiefly in

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the neighborhood of the disk, but also from other portions of the retina, consisting of pus-corpuscles and large nucleated round cells with granular contents in a net-work of branching fibres.

It is evident that the clinical picture constituting acute panophthalmitis is often dependent upon a purulent retinitis and cyclitis rather than upon a purulent chorioiditis, and this is often seen on making an equatorial section of the globe, when the posterior half of the sclerotic with the corresponding portion of the intact chorioid comes clean away, leaving the solidified vitreous surrounded by the thickened, inflamed, and adherent retina.

In examining eyes lost for many months or some years after panophthalmitis, especially of the less acute form, we find the chorioid and retina enormously expanded and forming one continuous mass, nearly filling the whole eye, and pervaded by a newly formed set of vessels. In places only can be seen the uveal pigment in clumps, forming an interrupted line, and indicating the level of separation of the chorioid and the retina, the former still recognizable by its branching pigment-cells, the latter retaining not a trace of its normal structure, but forming a true papillomatous growth with prominences on its inner surface. This may be termed the stage of carnification and hyperplasia, and has been well described by Schöbl. Plates of bone will often be found about the level of the uveal pigment remains, and the sclerotic is greatly thickened.

Prognosis.—Once panophthalmitis has started the eye is doomed, nor can we arrest the progress; but there is no danger to life in the traumatic cases, nor is sympathetic ophthalmitis likely to occur.

Treatment.—The indications for treatment are to alleviate the pain and give exit to the pus: this is done by the free use of poultices, followed by section through the sclerotic and well into the vitreous, between the external and inferior rectus tendons.

The aperture must be kept open by a drain, and the cavity daily syringed with a solution of perchloride of mercury of the strength of one in a thousand parts of water.

Enucleation of the eye cuts short all pain and discomfort, and allows of the patient's getting rid of his trouble in a few days, but is certainly not free from the danger of purulent meningitis and death: whilst this unfortunate result is relatively rare, a good many such cases have been recorded. This seems most likely to occur if the operation is done after the orbital tissues have become infiltrated, when enucleation is much more troublesome to perform on account of the thickening and adhesions of the extra-ocular tissues, and escape of pus, from perforation with the scissors or spontaneous giving way of the globe at some thinned portion, is apt to occur. It must be stated that it is the routine practice of some operators to enucleate in any stage of the disease. I have seen, but not in my own practice, two fatal cases of purulent meningitis and one case which recovered, and I cannot approve of this procedure, even if carried out with the strictest antiseptic precautions.

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It is claimed, and with some reason, that evisceration—*i.e.*, removal of the cornea with a ring of the surrounding sclerotic and complete clearing out of the contents of the globe—is to be preferred to enucleation in cases of this class, and that it is free from danger. It certainly leaves a better stump for an artificial eye.

COLLOID BODIES.

In phthisical eyeballs one often finds minute grayish hemispherical bodies projecting from the inner surface of the lamina vitrea and measuring from one-quarter to one-half millimetre in diameter. They are called colloid bodies, and are supposed to originate from a transformation of the protoplasm of the pigment epithelium of the retina; they may be hyaline or striated, frequently tend to become calcareous, and are often found in connection with bony formations in the chorioid.

The pigment epithelium is either raised over them or pushed aside and allows of their being seen. The chorioid may be so thickly studded with these bodies as to resemble a piece of shark-skin.

They occur as a senile change, but also in young persons, and then mostly in connection with inflammatory processes. As we shall see later, the presence of these colloid bodies gives rise to an ophthalmoscopic picture closely resembling disseminated chorioiditis.

BONY FORMATIONS OF THE CHORIOID.

Extensive formation of true bone in connection with the chorioid is not infrequently found in eyes that have been lost for many years, and we may recognize this condition, even before removal of the eye, by the peculiar feel such eyes give to the finger, as of a ball of wood covered with chamois leather. The mass of bone lies on the inner surface of the chorioid, and may form a complete cup, perforated behind by the stalk of the detached retina and open in front, the brim extending just as far as the true chorioid. I have seen one case in which an imperfect bony septum lay behind the lens and covered in the open end of the cup, but this is very uncommon.

A ring of bone is sometimes developed on the inner surface of the ciliary body; in other cases the bone takes the form of a ring around the optic disk; and sometimes all we find is a small, irregular plate at some part or other of the chorioid.

When the formation is coextensive with the chorioid, the outer surface of the bony shell will be found comparatively smooth and closely adherent to the chorioid, the inner surface being rough and shaggy-looking. It has throughout a brownish color.

The bone is thickest behind, often measuring several millimetres, and gradually becomes thinner as we approach the ciliary body. According to Brailey, the osseous deposit occurs mostly on the retinal surface of the lamina vitrea, and when, as rarely happens, it does occur within the chorioid, it extends no deeper than the chorio-capillaris.

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Microscopic sections show spicules of true bone arranged parallel to one another and to the surface of the chorioid, with trabecular tissue and fat-cells intervening and fine blood-vessels with capillary walls. No blood-vessels enter the bone itself. I have on several occasions found bone developed around a piece of metal in the eye.

It takes at least fifteen years to produce the large shell-like pieces of bone, but the small plates I have known to become developed within three years of the loss of the eye.

These bone-containing bulbs are often the sites of pain and tenderness, and may require enucleation on this account, but they are not liable to set up sympathetic ophthalmitis; indeed, I have never met with bone in eyes removed for this disease.

In sympathetic ophthalmitis the exciting eye, as described by Brailey, usually shows well-marked plastic uveitis, the iris, the ciliary body, and the chorioid being infiltrated with clusters of round cells, the chorioid often attaining a uniform thickness of one millimetre or more. Purulent infiltration is very rarely found in the exciting eye, a fact which is quite in accordance with one's clinical experience that panophthalmitis is so rarely a cause of sympathetic disease that it may be regarded almost as a safe-guard. I have found giant cells in the plastic chorioiditis affecting the exciting eye.

Metastatic Purulent Chorioiditis.—The causes of metastatic chorioiditis have been referred to already, and in this connection it will be necessary only to lay emphasis on the fact that the eye-affection is dependent upon a general septic condition of the system which is often fatal, and which is always of such gravity as to overshadow the local condition.

We must, however, say something in detail of two forms,—that occurring after childbirth, and the so-called pseudo-glioma of children.

Post-Partum Ophthalmitis.—The puerperal form occurs about the sixth day after delivery, and usually not later than the end of the second week.

It is often attended by a fatal issue, and when bilateral, in which case the second eye follows the other within a very few days at the most, it appears to be almost invariably fatal. The bilateral form is about half as frequent as the unilateral.

Loss of sight comes on rapidly, and is generally complete before the external signs of inflammation are at all advanced, there being, perhaps, only faint injection with iritic adhesions, slight hypopyon, and little or no pain. Even at this stage no fundus-reflex is obtainable.

This is followed in most cases by all the usual signs of acute panophthalmitis with perforation and escape of pus.

The disease sometimes runs a less acute course, the inflammation not extending to the orbital tissues, and perhaps admitting of an imperfect view of the fundus; but even in these cases the little sight retained for a brief period is always ultimately lost, and shrinking of the globe comes on in time, though without perforation.

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I have seen two monocular cases, coming on after normal confinements in healthy young women, that were characterized by the presence in the fundi of large, rounded, bluish-white, flocculent masses like packed cotton-wool. Neither showed external signs of inflammation. In one the mass was situated in the upper part of the eye, there was a limited separation of the retina stretching from the central region to the nasal side, and letters in 16 Jäger could be read. In the other case the posterior half or so of the vitreous cavity was occupied by the bluish-white, flocculent mass, with a denser white patch situated at its summit, the whole structure quivering like a jelly when the head was moved. The pupil was free and the iris acted well through the other eye, the tension was normal, and hand-movements could be recognized at the temporal side. When seen ten years later, the globe was shrunken, squared, and furrowed by the tendons of the recti muscles. It was quite soft. The cornea was clear, the anterior chamber was deep, and the pupil was occluded by a white capsular opacity. The iris-tissue was healthy, but presented a concave surface and showed posterior adhesions. There was no perception of light, and the eye was free from redness or tenderness. It appeared that the shrinking had followed a spontaneous inflammation of the eye some seven years before.

There is good reason for believing that the starting-point is due to septic embolism of the chorioidal or perhaps more frequently of the retinal vessels. Hosch has found bacterial thrombi in the retinal vessels, and Hirschberg has had the opportunity, in an early case, of seeing through the cloudy vitreous embolism and hemorrhages in the retina; the post-mortem examination confirming the nature and site of the starting-point in the retina.

Even ordinary cases of embolism of the central artery of the retina may be followed by suppurative panophthalmitis. I have seen one case, and J. Adams has recorded a case coming on two months after the arterial blockage.

In fatal cases examination often reveals recent ulcerative endocarditis with bacterial thrombi in the parametrium, kidneys, and other internal organs, with localized suppuration in various parts of the integument of the body.

Pseudo-glioma.—Pseudo-glioma is a term applied to certain cases of destructive ophthalmitis occurring in children under two years of age, characterized by a yellowish exudation behind the lens, and simulating real glioma of the retina.

The affection was binocular in eleven of twenty-seven cases collected by Nettleship.

When seen by the surgeon the eye is usually but slightly if at all injected, of lowered tension, and diminished in size, although the shape is well retained. The pupil is narrow and irregular from adhesions, the iris-tissue is muddy and opaque, the pupillary portion is advanced towards the cornea, while the ciliary zone is retracted and shows a circular furrow, the

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iris forming a truncated cone with the narrower end at the pupil, and presenting the appearance of having been moulded over the lens.

Immediately behind the clear lens is seen a layer of exudation, which gives a dirty-yellowish reflex never quite like the beautiful pure white or pinkish-white lustre of real glioma.

The history of a general illness, the injection, the iritic adhesions, the retraction of the periphery of the iris, the diminished tension, and the above-mentioned character of the reflex, should prevent such a condition from being mistaken for glioma.

I have known pseudo-glioma to occur coincidently with and mask the presence of real glioma: in this case the eye became glaucomatous and was enucleated.

In some cases, if seen early, we find marked injection of the eye and pus in the anterior chamber; but these acute symptoms are exceptional, do not persist long, and never go on to perforation.

After the subsidence of the acute symptoms the eye gives no trouble, and, save for opacity of the lens sometimes coming on, remains unchanged.

The sight is, of course, always completely destroyed. The inflammation, whatever its exact nature, is probably conveyed to the eye through the lymph-channels. I am not aware of any bacteriological examinations having been made in this class of cases.

In all cases we find a puro-plastic exudation covering the ciliary body and the back of the lens, and the latter is often distorted, its posterior surface assuming a conical shape, the result, no doubt, of traction produced by the shrinking of the exudation. The retina is most commonly completely detached, forming a round cord behind, which in front expands in a corolla-like form, the folds being thickened and agglutinated by lymph.

A net-work of easily stained homogeneous tissue, sometimes with collections of blood, stretches from the chorioid to the detached retina, filling up the space between these structures. In a smaller number of cases the retina is not detached, but is thickened, and long strings of lymph stretch from the exudation at the back of the lens to the optic disk and its neighborhood.

The chorioid is usually apparently quite unchanged, but is sometimes thickened, and may contain one or more very large extravasations of blood. The affection may occur in the course of measles, varicella, bronchitis, very often in syphilitic infants, in whooping-cough, cerebro-spinal meningitis, etc., or without any known form of disease; but in any case the eye-symptoms are usually immediately preceded by symptoms suggestive of meningitis, as great pain in the head, rise of temperature, unconsciousness, spasms, and temporary weakness of muscles.

In one case I saw, the medical attendant, who was a competent observer, noted, besides the pain in the head and unconsciousness, contraction of the sterno-mastoid muscles and hyperpyrexia for over three weeks, the temperature rising to 106° Fahrenheit.

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As far as I know, the symptoms subside rapidly and permanently, death never takes place, and the patients recover their health completely.

No treatment is required except the use of atropine, if seen early; subsequently the eye gives no trouble, and it is very seldom that enucleation is called for.

Congenital Irido-Chorioiditis.—A congenital form of irido-chorioiditis, due to intra-uterine inflammation affecting one or both eyes, is sometimes met with in badly nourished and mentally deficient infants of tubercular or, less commonly, syphilitic diathesis. I saw an example in an otherwise healthy infant of seven months, with strongly tubercular family history, in which the right eyeball was small, but free from injection, and the eyelids hung in a flaccid manner characteristic of such a condition. The iris-tissue seemed healthy, but the pupillary edge was adherent to the lens-capsule, and the latter was covered with false membrane, so that no fundus-reflex could be obtained.

When the condition of the media permits the use of the ophthalmoscope, a yellowish reflex is obtained from the depth of the eye.

Plastic Chorioiditis.—The various forms of lesions under this heading are discernible only by the use of the ophthalmoscope: hence it will be necessary before discussing them to describe the normal ophthalmoscopic appearances of the chorioid.

Ophthalmoscopic Appearances of the Normal Chorioid.—A large portion of the chorioid cannot be seen at all by the ophthalmoscope,—namely, all that lies anterior to the equator of the eye,—and the appearances presented by the remainder vary according to the amount of pigment in the retina and the chorioidal stroma.

If the pigment in both these situations is abundant, as in persons of dark hair and complexion, we get a uniformly brick-red or brownish-red background without a trace of chorioidal vessels, and with the greater magnification obtained by the direct ophthalmoscopic examination this surface appears stippled.

When the pigment is absent in the retina but present in the chorioid, we get what may be termed the "tiger-skin" fundus, the larger chorioidal vessels appearing as curved red lines and the intervascular spaces as darker brown areas.

In very fair persons, where there is little or no pigment in both retina and chorioid, we have the "albinotic" type of fundus, the entire system of chorioidal vessels being visible as far as the magnification will allow, the interspaces appearing quite pale on account of the sclerotic shining through the transparent structures.

Intermediate types of fundi are of course met with, and the same eye often shows more or less marked difference, the chorioidal vessels being more exposed towards the periphery than at the central region, where the retinal pigment is more abundant and the chorioid thicker.

Apart from the question of the complexion, the chorioidal vessels are

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always more visible in myopia than in other states of refraction, and in old people than in young.

In cases of marked astigmatism the portion of chorioid corresponding to the meridian of greater curvature is usually characterized by more or less exposure of its vessels: this is especially noticeable in the myopic meridian of mixed astigmatism.

Chorioidal vessels are distinguished ophthalmoscopically from retinal vessels by their flat, tape-like form, their light red, uniform color and the absence of a central streak, their great tortuosity, and the manner in which the larger trunks break up into a number of branches at one point, like a piece of sea-tangle. Where crossed by the retinal vessels, the vessels of the chorioid can be seen to lie on a posterior plane. No distinction can be made between veins and arteries, although, as we have seen, this is easily done in anatomical examination. Their course becomes much straighter as we near the equator, where they join to form the four or five vorticose veins.

Hyperæmia of the Chorioid.—Hyperæmia of the chorioid cannot be diagnosed with certainty, and we are unacquainted with the symptoms, if indeed there are any, to which such a condition gives rise. The only ophthalmoscopic sign is increased vascularity of the disk, the deeper vessels of which anastomose with those of the chorioid.

Even patches of localized inflammation may not be discoverable till they have broken through the hexagonal pigment-layer of the retina, especially if there be much pigment in this layer.

In most ophthalmoscopic forms of chorioiditis the changes certainly start in the inner layers of the chorioid and speedily implicate the uveal pigment and bacillary layer of the overlying retina, the pathological changes often going no further than this: hence the term chorio-retinitis is often a more suitable name than simply chorioiditis.

We shall consider plastic chorioiditis according to the following classification:

(1) Disseminated chorioiditis; (2) syphilitic chorio-retinitis; (3) central chorioiditis; (4) posterior sclero-chorioiditis.

Disseminated Chorioiditis.—In chorioiditis disseminata the changes may be entirely confined to one eye, or they may affect both, in which case one eye is often much more extensively implicated than the other.

However it starts, and no matter how slight, disseminated chorioiditis always leaves permanent patches of atrophy, which vary in appearance chiefly according to the depth to which the process has penetrated the chorioid. The changes may be pretty evenly distributed all over the fundus or confined to a limited area.

Patches of complete atrophy appear as rounded areas from one-quarter to one disk's diameter, or a little larger, bounded by a more or less complete ring of black pigment; the entire surface within this ring is white or bluish-white and glistening, and shows no trace of chorioidal vessels or pigment; the retinal vessels course uninterruptedly across it. The black

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ring is due to a crowding aside and proliferation of the retinal pigment, and the enclosed white area corresponds to the inner surface of the sclerotic, laid bare by the atrophy and disappearance of the chorioid.

The appearance of the fundus between the atrophic spots is usually normal.

In other places we may find similar patches showing tiny splashes of pigment on the exposed sclerotic, probably the remains of the lamina fusca; in other patches, again, are seen the chorioidal vessels, either quite unchanged, as if dissected out, or with partial or complete obliteration of the blood-stream from endarteritis, appearing as white or yellowish-white strands, some with, others without, a narrow red streak in their centre.

More or less of the pigmented chorioidal stroma may persist at the affected spots, in which case the spaces between the chorioidal vessels appear brown, and the vessels themselves in places may be partially veiled by the presence of more or less of the retinal pigment.

The amount of pigment at the margins of these atrophic patches varies from a mere hair-line to quite a massive black ring, and depends chiefly upon the amount of pigment present in the hexagonal pigment-layer before the onset of the affection, and to some extent upon the occurrence of hemorrhage.

At the diseased spots the outer layers of the retina are always more or less infiltrated with the pigment, which may even penetrate to the nerve-fibre layer and coat the retinal vessels.

The outer layers of the retina become quite atrophic and merged in the chorioidal cicatrix; the nerve-fibre layer and its vessels, as a rule, show little if any change.

Early Stage.—Under favorable circumstances we may study the changes which any particular spot undergoes from the day it first reveals its presence to its final stage as one of the above-described atrophic patches.

It is first seen as a rounded or perfectly circular white spot with a delicate pink tinge and semi-translucent appearance; the edges are softened and gradually shade off into the surrounding fundus.

Any retinal vessels are much more clearly defined where they cross the surface of the spot, and if very minute may be nearly or quite invisible beyond its borders.

This stage depends upon the presence of a layer of exudation on the inner surface of the vitreous lamina, which has broken through the uveal pigment-layer and overrun its edge, so that no pigment whatever is to be seen. One cannot make out any elevation of surface.

These initial changes are sometimes partly or completely concealed by a chorioidal hemorrhage, distinguished from the surrounding surface by its uniform deep-red color.

The next change takes place at the centre of the spot, which becomes very white and opaque, like a spot of thick white paint, and about the same time the pigment-ring commences to show itself like a dark shadow,

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gradually becoming more pronounced, and reminding one of the development of a photographic negative.

The pigment-ring and other details now sharpen up, and the enclosed surface appears as a uniform dull yellowish-white plaque, concealing the underlying chorioidal vessels, and in this way being distinguished from the later atrophic stages already described.

Little shreds or films of lymph are often to be seen at this stage attached by one edge to the chorioidal plaque and fluttering in the vitreous; also little, shining spangles of cholesterin which appear as if actually incandescent, and clusters of white, silvery-like flecks.

Vitreous opacities are often absent through the entire course of the disease. Their presence depends upon the coincident implication of the retina rather than upon the chorioiditis itself, and they are found only when the retinitis is more marked than usual and the inner layers are affected, as shown by opacity of the retina and blurring of its vessels. The retinitis in this case is to be regarded as a separate process, brought about by the same general cause as the chorioiditis, and not simply as a part of or an extension from the latter.

When the inner layers of the retina do become affected the case is always much more serious. The veins become engorged, the disk and its neighborhood are opaque and hazy, and long strands of lymph proceed from this region towards the ciliary body. Total loss of sight may take place, either from atrophy of the disk or from detachment of the retina, followed by complete opacity of the lens, sometimes with, sometimes without, the intervention of iritis. Iritis, interstitial keratitis, and scleritis sometimes make their appearance in the course of the disease.

In old-standing cases of chorioidal atrophy liquefaction of the vitreous may take place, with partial or complete dislocation of the lens, which may fall back and be found lying at the floor of the vitreous cavity, or may become displaced through the pupil into the anterior chamber and set up acute glaucoma, as I have seen on several occasions.

In addition to the disseminated spots of chorioiditis, one often finds at some part of the fundus, and especially about the central region, quite extensive plates of pigment, surrounded by a zone of yellowish discoloration the outer edge of which closely approximates in shape that of the pigmented plate, as if the latter had undergone some shrinking. The affected areas vary greatly in shape, being stripe-like, serpiginous, approximately square with incurved sides, or of quite fantastic form; they are continuous one with another by narrow processes, or form detached foci, and one often sees the larger pigmented plates cracked across the centre. The appearance in these cases, as suggested by Michel, is as if the fundus had been overrun with some corrosive fluid.

Sometimes the disease differs considerably from any of the above-described varieties, appearing as small, irregularly shaped but sharply defined, dirty-yellowish spots without any pigmentation and with no exposure of

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the chorioidal vessels. They look as if the uveal layer had been very gently scraped away with the point of a sharp knife.

Areolar Chorioiditis.—Förster long ago described a variety of chorioiditis which he termed *areolar chorioiditis.* It is probably not an inflammatory condition; it occurs nearly always in both eyes; the changes are confined to the region of the disk and macula, and consist of round, sharply defined, punched-out patches of atrophy of the chorioid, rather larger than the optic disk, always very closely set together, and sometimes coalescing. The atrophy of the chorioid within the affected patches is usually complete, showing the bluish-white, tendinous-looking sclerotic. There is often a very narrow border of coal-black pigment.

If seen early, which is not often the case, the patches are nearly uniformly black and become decolorized from the centre.

Central vision often fails suddenly about the fortieth year of life without further ophthalmoscopic changes.

Symptoms of Disseminated Chorioiditis.—The ophthalmoscopic appearances of disseminated chorioiditis are very easily made out, and are liable to be passed over only if, as not infrequently happens, they are confined to the extreme limits of the visible chorioid. Although for the purpose of a bare diagnosis the subjective symptoms are of secondary importance to the ophthalmoscopic signs, still it is always advisable to make repeated examinations of the vision and the field of vision, in order to ascertain the progress of the affection and its probable termination.

The symptoms mainly depend upon the implication of the percipient elements of the retina, and a careful functional examination is required to determine the extent of this.

When the changes are confined to the periphery there may be no symptoms and no defect of sight, and the disease may be revealed only by an ophthalmoscopic examination undertaken for correction of refraction or for some other purpose.

Disproportion between the condition of the chorioid and the state of the vision is a common and striking feature. The chorioid may be absolutely riddled with disease while the central vision is little if at all affected, and the latter may rapidly fail without any fresh ophthalmoscopic signs.

The patients usually complain of clouds of black specks, flickerings, flashes of light and colors, or of a cloud in front of the sight, and often also of discomfort or even pain around the orbit.

The central vision is nearly always lowered. Fine print can be read, if at all, only in bright daylight.

In cases of recent exudation about the macula there are often present two other symptoms, *micropsia* and *metamorphopsia*, which are not commonly spontaneously complained of by the patient, and are best made out by causing him to look at a set of parallel lines drawn on a surface near at hand, when the central ones will appear to him as if bent towards the fixing-point, showing both the presence of diminution in size of the object

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(micropsia) and distortion of its outlines (metamorphopsia), conditions due to displacement and separation of the percipient elements of the retina by the chorioidal exudation.

The opposite condition of *macropsia*, due to overlapping of the retinal elements, occurs during the shrinking of the exudation, and is shown by a bulging outward of the lines from the fixing-point.

The field of vision may show an absolute scotoma, either as a central black spot or as a ring around the fixing-point. The periphery may be of normal extent, or may show contraction or a sector-like defect at some part, and one can nearly always demonstrate the presence of several scotomata between the centre and the periphery of the field.

These scotomata are spoken of as *positive* when the patient is conscious of their presence as dark or black spots, and *negative* when perimetric examination is necessary for their demonstration.

When objects within their area completely disappear, the scotomata are called *absolute*, and when only indistinct, *relative*. The examination should be undertaken in a subdued light.

Causes.—Apart from trauma, the disease is brought about by micro-organisms conveyed to the eye from within the body by the circulation of the blood, and these organisms must be arrested and adhere to the walls of the vessels before they can cause damage, such arrest being commonly promoted by their being contained in coagula or particles of broken-down tissue, or aggregated in colonies, whereby they are rendered incapable of traversing the finest arterioles or capillaries.

Syphilis, more commonly the acquired form, and tuberculosis are certainly by far the commonest causes of disseminated chorioiditis, but rheumatism, gonorrhœal infection, especially when the joints are implicated, bad conditions of nutrition, or poorness of blood without special cause, severe forms of malaria, and profound anæmia, must all be reckoned as more or less frequent causes of the affection. Osteitis deformans, or Paget's disease, typhus, and typhoid fever have been given as probable causes in some few recorded cases.

In a not inconsiderable, perhaps in what may be termed a large, proportion of cases no cause can be assigned.

It is considered by some authorities, and my own experience bears it out, that tuberculosis is almost if not quite as common a cause as syphilis.

The occurrence of a group of yellowish or white flecks near the macula, and of dust-like opacities in the vitreous, with changes in the walls of the chorioidal and retinal vessels, points very strongly to a syphilitic origin. The tubercular cases, besides differing in these points, occur more frequently in one eye. The changes are not so widely spread, pigmentation is not so marked a feature, and, perhaps most important of all, the patches undergo atrophy much more slowly.

In acquired syphilis the chorioidal disease makes its appearance from six to eighteen months or more after infection; in congenital syphilis some-

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times at birth, but more frequently at the age of from six months to three years, and later it is often combined with the characteristic interstitial keratitis, and two or more members of a family may be affected.

The determination of the cause, however, will depend much more often upon the history and the general examination of the patient than upon the special characteristics of the eye-affection.

Treatment.—If seen early, all cases should be absolutely forbidden to use the eyes for close work or to expose them to a bright light, and this is best insured by the use of atropine and dark-smoked protective spectacles.

If any signs of syphilis are present, or if we get a clear history of syphilitic infection or symptoms, a prolonged but mild mercurial course is indicated, usually by the internal administration of mercurial pill, the perchloride, gray powder, or other preparation, reserving inunction—the mode of using which will be fully described under Syphilitic Chorio-Retinitis—for cases in which deterioration of vision is taking place rapidly.

The general health must not be neglected. Fresh air, good food, and tonics form an important part of the treatment, and in debilitated, badly fed dispensary patients a few weeks' residence in the hospital, with liberal diet, will often work wonders.

When occurring in syphilis of old standing, some prefer the iodide of potassium. Personally I have much more faith in mercury: when I order the iodide it is only when the mercury has had to be stopped on account of the state of the gums, and I never give it in doses of less than ten or fifteen grains.

Even after all changes in the fundi have apparently ceased for some time, the vision may again fail, with flashes of light, etc., and under such circumstances we must again have recourse to active treatment.

The good effects of mercury in syphilitic cases is a matter of common observation and beyond all doubt; but when this disease can be positively excluded it does seem unreasonable to limit one's treatment, as some do, exclusively to antisyphilitic remedies. In such cases a little enterprise and observation of the effects of other remedies would, I am sure, do no harm to the patients, and might put us in possession of some useful information in regard to the non-syphilitic forms of the disease, the treatment of which is admittedly unsatisfactory. Cod-liver oil, the hypophosphites, preparations of iron, iodoform, or, better, creosote, certainly do good in tubercular cases, and salicylate of sodium, arsenic, or colchicum would probably be useful when the general condition indicated their employment.

The prognosis is decidedly better in the syphilitic cases. Good and sometimes normal sight may be regained; and from a large number of observations I am prepared to state that the vision so regained is, in the large majority of such persons, permanently retained, and permits them to follow their employment successfully.

Colloid Degeneration.—There is a class of cases which may be best described here, as the ophthalmoscopic appearances bear a close resemblance

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to true disseminated chorioiditis, but are certainly in most if not all of the cases dependent upon the presence on the inner surface of the vitreous lamina of a number of those minute excrescences which have been variously described as chorioidal verrucosities, colloid excrescences, colloid bodies, hyaline bodies, etc.

These colloid bodies, as mentioned before, are often found in lost and disorganized eyes, especially in connection with bony formations. They are also accountable for some of the appearances in various forms of chorioiditis; but in the group of cases under discussion they seem to represent the sole pathological appearances.

In most of the published cases, and in all that I have myself seen, the changes were symmetrical. The vision, both central and peripheral, was normal, and these conditions remained unchanged throughout.

The affected area extends from a little to the nasal side of the disk to the equatorial region at the temporal side, and the fundus within this area is covered, and most thickly about the macula, with round, dense, white, chalk-like, discrete spots about the diameter of the retinal vessels. The spots may to some extent coalesce at the macula, forming mulberry-like masses. They may in some cases be yellowish or gray rather than white, and some of them may show a trace of pigment at their margin. This condition is usually met with in young adults, and in some cases is no doubt congenital.

No treatment is required.

Syphilitic Chorio-Retinitis.—Syphilitic chorio-retinitis is always characterized at first by fine dust-like vitreous opacities and increased redness of the disk, which is surrounded by a halo of grayish discoloration.

Before the publication of Förster's investigations this affection used to be called syphilitic retinitis, a term which some still employ; but it is generally conceded that it ought to be regarded as a form of superficial inflammation of the chorioid.

The absence of the usual patchy condition characteristic of chorioidal as distinguished from retinal inflammation is probably due to the microbic irritant being in solution.

It is almost invariably caused by syphilis. It comes on from six to eighteen months after infection, much more frequently affects both eyes, is very slow in its progress, and is much more severe when it occurs in persons beyond middle life. It is not infrequently preceded by iritis, as shown by the presence of posterior synechiæ.

At first the acuteness of vision may be only slightly diminished, but night-blindness is usually a marked feature, and the patient may have the greatest difficulty in walking out of doors, often injuring himself by knocking against objects in his path. Micropsia and flashes of light are also commonly present.

The field of vision seldom shows any diminution in extent, but scotomata are often present.

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Variations in the power of sight are frequent and marked, and this is explained by the great tendency for different parts of the vitreous to clear up and again become clouded.

In applying the tests for acuteness of vision plenty of time should be allowed, for it will often be found that after a minute or two a higher standard is reached. We must also be prepared, in testing the near vision, to find some weakness of the accommodation and neutralize this by convex glasses.

In early and slightly marked cases the diagnosis is often exceedingly difficult. The "smoky" appearance of the details of the fundus so characteristic of the disease may also be caused by a faint central nebula of the cornea, especially in examination by the direct method, and we must always exclude this condition by careful focal illumination. This form of chorio-retinitis not infrequently occurs with patches of ordinary chorioiditis, and if these are few in number and far forward we may easily overlook them. In elderly persons the disk and its neighborhood are usually less clear than in young adults, and when this is more marked than usual we may erroneously diagnose specific chorio-retinitis. The ophthalmoscopic signs are less definite and more easily overlooked than those of any other disease in the fundus.

The vitreous opacities, increased redness of the disk, and opacity of the surrounding retina may persist for many months as the sole ophthalmoscopic signs of the disease, and under treatment a perfect recovery often takes place at this stage, so that it might be impossible to say that the eye had ever been other than normal.

Later, if the disease advances, there is often found overfulness of the retinal veins, with diminution in calibre of the arteries and yellowish discoloration and paleness of the disk.

The dust-like opacities clear away to some extent, their place being taken by larger black opacities, which by their free mobility indicate some fluidity of the vitreous.

In cases of long standing, especially in old people and when treatment has not been properly carried out, still further changes occur. Towards the periphery more or less extensive and irregular but sharply defined tracts are met with in which the retinal pigment has disappeared, exposing the chorioidal vessels, with angular and scrappy pieces of pigment infiltrating the overlying retina and coating its vessels in places. The central region is sometimes occupied by a large, dense, bluish-gray opacity, with numerous cicatricial bands intersecting its surface like a piece of coarse matting.

At this stage the vision is almost if not completely lost, not from atrophy of the optic nerve, for the disk is often fairly healthy in appearance, but from degeneration of the retina.

In some cases of very old standing we find the retinal pigment entirely wanting all over the fundus and the chorioidal system of vessels fully ex-

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posed to view, just like the physiological "tiger-skin" fundus, from which it is distinguished ophthalmoscopically by the presence of thickening or other changes in the walls of the chorioidal vessels, and by the occurrence of fragments of coal-black pigment in the vascular layers of the retina, in some places coating the retinal vessels, in other places taking the form of little bundles of fagots.

This condition might on a superficial examination be mistaken for true *retinitis pigmentosa*, especially if, as sometimes happens, there be a stellate opacity at the posterior pole of the lens; but in retinitis pigmentosa there is usually no exposure of the chorioidal vessels, the pigment is of a more delicate lace-like or mossy character and coats the retinal vessels much more extensively, the field of vision always shows regular peripheral contraction, and central vision is usually good, and always much better than the peripheral vision.

This condition is possibly always merely a late stage of syphilitic chorioretinitis, but it occurs at such a long time after the commencement of the original affection that we can seldom verify this by actual observation.

Treatment.—The best treatment is that by mercurial inunction, and, when the patient can be kept under the surgeon's observation, this method of using mercury should always be employed.

The first thing to do is to make the patient pay a visit to the dentist to have all collections of tartar scaled off the teeth, sharp angles filed down smooth, and decayed stumps removed.

One or two drachms of mercurial ointment are to be rubbed into the skin of the axillæ or inner side of the thighs each night, and after every meal the toothpick is to be used and powdered alum applied to the gums.

If these precautions are attended to we need have no fear of mercurialization, and we can carry on the treatment for many weeks without producing anything more unpleasant than slight redness and sponginess of the gums, and often without even this.

Under this treatment we may get a surprising increase of vision, even, as I have seen, from 20 Jäger to 1 and 6/6.

Central Chorioiditis.—When chorioiditis is confined to the region of the macula it is spoken of as *central chorioiditis*, and in this form may occur as a senile change, or in young persons often without special cause.

Slight central changes can often be detected after blows on the eye, embolism of the central artery of the retina after intense optic neuritis, in high myopia, and in tumors of the orbit or orbital phlegmon, but these cases will not be considered here.

I saw two young persons, brother and sister, the latter of whom had typical symmetrical central chorioiditis, the former ordinary chorioiditis disseminata. These and a few other observations of the kind indicate that the disease in question may sometimes be a limited form of the ordinary disseminated chorioiditis; but this is true only in a small proportion of cases, the majority being of a totally distinct nature.

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The senile form is nearly always symmetrical, and very seldom makes its appearance before the sixtieth year,—usually, indeed, at a later age; the central vision is always reduced, often to 16 or even 20 Jäger; and although total blindness never occurs, no improvement whatever is to be expected.

It is by no means an uncommon affection, and should always be searched for in cases of incipient cataract, as its presence would greatly modify our prognosis as to restoration of sight by operation at a later stage.

It must also be kept in view in all cases of gradual loss of reading power in old people with normal fields and transparent media.

In its typical and well-developed form the disease has the appearance of a circular or slightly oval, sharply defined spot at the macula, about the size of the disk. It is paler than and lacks the lustre of the surrounding fundus, and its surface shows yellowish erosion-like dots and pigmentary disturbance; sometimes also minute punctiform hemorrhages and crystals of cholesterin. In many cases the changes are slight, perhaps only an irregular cluster of yellowish dots at the macula, or an appearance as if this region had been lightly dusted over with a mixture of salt and black pepper.

In course of time the atrophy of the chorioid becomes more marked, but it is never complete and never extends outside the limits of the area originally affected.

Similar appearances are occasionally met with in quite young people, and, so far as my experience goes, are more often associated with anæmia than with any other condition; and considerable improvement in sight often takes place under treatment.

If careful and systematic examination of the macular region be carried out as a routine practice, we shall not infrequently meet with slight chorioidal disturbance when the vision is quite normal, or when tobacco amblyopia, uncorrected refraction, or some condition other than the chorioidal disease is the cause of the defective sight.

I have on several occasions seen both the senile and the non-senile form in two or more members of one family, and others have also noticed this point.

Anomalous Forms.—We shall here describe some anomalous forms of chorioiditis.

I saw with a medical friend a young nephew of his whose sight for reading had always been rather defective, and I found that this was accounted for by the presence of an irregular mass of black pigment at the central region of each eye, with slight thinning of a narrow strip of chorioid around. Syphilis and tubercle could be definitely excluded, and I concluded that the damage had taken place at his birth, for the labor was an excessively long and difficult one, accompanied by enormous muscular exertion on the part of the mother. I had previously seen some other cases of the kind.

Chorioidal "Craters."—We sometimes meet with a single large area of chorioidal atrophy at or near the centre of an otherwise normal eye which

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has the following characters. The area is several times the size of the disk, nearly circular, and sharply defined by a well-marked ledge; the sclerotic within the affected area is strongly bulged backward; the chorioidal vessels are destroyed, or more commonly freely exposed, with large masses of coal-black pigment between them, reminding one of the appearance of the web of a frog's foot under the microscope; and the retinal vessels are free from disease. The changes are quite stationary; one or both eyes may be affected. If, as is nearly always the case, the macula be implicated, vision may not reach higher than 20 Jäger, or even only "fingers;" but I have seen the vision practically normal.

When monocular, the eye is usually divergent.

Although the condition is probably in most cases present at birth, and occurs in healthy persons, I believe it is incorrect to call it, as some do, a coloboma of the chorioid. I have once or twice seen apparently similar changes begin to show themselves in the second eye later in life.

Chorioiditis with Descemetitis.—At the Ophthalmological Society of the United Kingdom, some eight years ago, I called attention to cases of solitary patches of chorioido-retinitis with descemetitis, and subsequent experience has confirmed me in the opinions I then expressed.

The disease is ushered in by sudden dimness of vision in one eye, which shows, on close examination, a greater or less number of very fine, gray, dust-like opacities on Descemet's membrane. There is complete absence of all external signs of inflammation, the iris is healthy, and the pupil is normal and quite active.

In the neighborhood of the disk is found a single, recent, opaque white or bluish-gray patch of chorioido-retinitis, shading off gradually at its circumference into the healthy fundus.

Very rarely there may be two patches close together and evidently of similar duration, or a recent patch may be continuous with an old pigmented spot, in which case we may elicit a clear history of an apparently similar attack years before. Fine vitreous opacities are sometimes seen near the chorioidal disease, or a long string of lymph may extend into the vitreous from the edge of the patch. If the chorioidal patch is continuous with the disk, the real condition may for a time be masked by a papillo-retinitis. The dots on the back of the cornea fade away rather quickly, the chorioidal patch becomes converted into an atrophic area, with exposure of the chorioidal vessels and pigmentation, and normal vision is frequently regained.

I hold that the dots on Descemet's membrane are in these cases formed in the chorioid, set free in the vitreous, and carried by the nutrient currents of the eye to be deposited on the back of the cornea, which view necessitates the permeability of the suspensory ligament by solid particles.

The patients have all been young adults, the female sex largely preponderating. I have seen between forty and fifty cases; syphilis has never been present, so far as I could make out, although ozæna has not infre-

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quently been noted; tuberculosis or a tubercular family history is very common, and my treatment has been based accordingly.

Posterior Sclero-Chorioiditis.—This is an inflammatory or degenerative process which produces a localized atrophy of the chorioid adjacent to the disk, with bulging backward of the sclerotic, a condition known as *posterior staphyloma.*

Extensive posterior staphyloma is the cause of the congenital malignant form of myopia, but it is also common in the ordinary form of myopia, especially in the higher grades.

It occurs as a crescentic area of atrophy of the chorioid, with its greatest diameter extending from the temporal border of the disk nearly as far as, and occasionally involving, the macula. It may, besides, completely encircle the disk by a narrower ring-shaped extension, and in some cases the atrophic area is dumb-bell-shaped, the portion around the disk being joined to that at the macula by a more or less constricted neck.

The affected area is bounded by a more or less complete narrow black ring of pigment, and within this various degrees of atrophy of the chorioid are met with, the most complete being next to the temporal side of the disk, where the sclerotic is completely exposed and has the appearance of a bluish-white, tendinous crescent. Outside this and concentric to it are other crescentic areas of diminishing degrees of atrophy, faint dividing lines indicating their formation at different periods of time.

The bulging of the coats of the eye is certainly most marked, *not* at the macula, but just beyond the temporal border of the disk, which is thus tilted backward, causing the transverse diameter to appear much less than the vertical; this effect being increased, as Weiss has shown, by the tissues of the nasal half being dragged over towards the temporal side.

When the changes are progressive we may meet with small splashes of chorioidal hemorrhage within the affected area, with opacities in the posterior part of the vitreous, and the vitreous is said to become detached from the staphyloma by a very thin fluid.

The exact anatomy of this condition is more fully described under the articles on Errors of Refraction and Staphyloma.

As mentioned before, the staphyloma may reach some distance beyond the macula, forming a large area from six to eight times the size of the disk; but in high myopia, even when it does not reach the macula, changes, as spots of chorioidal hemorrhage or pigment, limited rounded spots of atrophy, or clusters of short white intersecting lines like cracked Bohemian glassware,—no doubt minute fissures in the chorioid,—are common in this region. Under these conditions central vision may be much reduced. As the greatest bulging does not take place at the macula, enlargement of the staphyloma is not accompanied by a corresponding amount of increase in the myopia. The latter may increase without any increase of the former, or even without the presence of any staphyloma.

When the staphyloma appears simply as a narrow, sharply defined

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white crescent, as it frequently does in low degrees of myopia, it is probably not dependent upon atrophy of the chorioid, but due simply to exposure of the sclerotic from the chorioid failing to extend as far as the disk, and it is impossible to make out any bulging whatever.

In rare cases the crescent may be confined to the nasal border of the disk. In astigmatism we often find it bounding the lower margin, the widest part corresponding to the meridian of greatest corneal curvature. I do not recollect ever seeing one limited to the upper margin.

A non-progressive but apparently similar small crescent is seen in a few emmetropic or even hypermetropic eyes.

Ring-shaped atrophy of the chorioid around the disk occurs as a purely senile change in all states of refraction. Thaden has shown that each decade of ten years beyond the age of fifty gives about double the proportion of staphyloma of the preceding decade, and that the atrophic area increases in extent as age advances. He found that seventy-two per cent. of persons over the age of eighty showed this condition. The atrophy in these cases is, unlike the crescent of myopia, usually incomplete, with exposure of the chorioidal vessels and pigmentation.

In glaucoma a ring of complete atrophy is often found immediately surrounding the excavated disk, the line of junction with the healthy chorioid being usually rather wavy but sharply defined.

RUPTURE OF THE CHORIOID.

Rupture of the chorioid is of rather frequent occurrence, and is produced by sudden compression of the bulb, usually from a smart blow on the eyeball by some hard body.

If seen within a day or two of the accident it is usually impossible to make out the lesion in the chorioid, on account of the presence of blood in the vitreous and cloudiness of the retina. Separation of some portion of the iris from its ciliary attachment, or rupture of its pupillary border with blood in the anterior chamber, is commonly present, and partial dislocation of the lens is at times found. In the course of a day or two, when the media have cleared, the rupture is seen as a yellowish-white crescentic streak at the temporal side of the disk, between it and the macula, with the concavity presented to the disk. The retinal vessels course uninterruptedly across it. At its central and widest part it is usually about one-fifth the diameter of the disk; it tapers off gradually to a fine point at each extremity, and extends for some little distance above and below the disk. The margins of the rupture are always more or less pigmented.

It is very exceptionally located at the nasal side of the disk. One or both extremities may be bifurcated, or two or more concentric ruptures may occur.

These characteristic crescentic ruptures are probably caused in the manner suggested by Becker: the optic nerve at the moment of the blow is driven into the sclerotic, producing concentrically to the disk a fold or

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series of folds in the chorioid and the retina, along which the former gives way.

When the blood has been absorbed and the retinitis has disappeared the vision may be partially or completely regained, but may afterwards be lost by separation of the retina during the period of cicatrization.

There is often left permanent dilatation of the pupil with loss of direct and consensual reaction, but this is not necessarily accompanied by loss of accommodation.

Atrophy of the disk may take place in severe cases. As the loss of sight precedes the pallor, this is probably produced by mechanical injury of the nerve at the time of the accident. Coarse black stippling of the macular region is often met with.

The lesion in the chorioid following a blow on the eye does not always assume the above-described form, but may occur as one or several irregular areas about the macular region, and, though none the less a rupture, cannot be explained on Becker's theory.

J. Hutchinson, Jr., and Little, at a meeting of the Ophthalmological Society of the United Kingdom, mentioned cases in which the pigmentary changes around the rupture went on progressing for long periods of time and extended over a very wide area, producing an appearance very like that of ordinary disseminated chorioiditis.

I have seen several cases in which the ruptures were partially or completely concealed by large masses of lymph which extended forward into the vitreous like loosely packed cotton wool.

CHORIOIDAL HEMORRHAGE.

Hemorrhages recognized by the ophthalmoscope are incomparably less frequently to be referred to the chorioidal than to the retinal vessels, and when they do occur in the chorioid they are nearly always an accompaniment to some other process in that structure, such as disseminated chorioiditis or progressive posterior staphyloma, and not, as in the retina, the sole recognizable change. When retinal hemorrhages occur, as they usually do, in the nerve-fibre layer, they are easily distinguished from chorioidal hemorrhages by their characteristic flame shape, and by their relation to the retinal vessels, the actual rupture of which can sometimes be seen; but when they are situated in the deeper layers it is impossible to say whether they are in the chorioid or in the retina. Chorioidal hemorrhages may be seen to lie *behind* the retinal vessels; their margins are rounded, and their edge is sometimes sharply defined, or gradually shaded off into the healthy chorioid; they are of a uniform rose color, never, like retinal hemorrhages, almost black, and during absorption pigmentation is met with in the chorioid.

Recent chorioidal hemorrhages, covering, as they often do, a very extensive area, and not differing much in color from the surrounding fundus, may easily be passed over, but with care one should distinguish the deeper red of the affected portion of the chorioid.

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Large chorioidal hemorrhage sometimes follows extraction of cataract in old people, leading to protrusion of the contents and loss of the eye.

Blows on the eye sometimes cause such a hemorrhage. Most cases of hemorrhage in the vitreous in young people are chorioidal in origin. Malaria is also a recognized cause of this condition, and after absorption of the blood one finds equatorial chorioiditis.

Although rare as an ophthalmoscopic condition, chorioidal hemorrhage is frequently found in lost eyes excised for glaucoma, fibro-purulent chorioiditis, intra-ocular growths, etc. In a fatal case of leukæmia, Oeller found the chorioid enormously thickened and infiltrated with lymph-cells, the result probably of an old hemorrhage.

Staphylomatous eyes excised for pain and increase of tension usually show extensive chorioidal hemorrhage, and under such circumstances I have seen the entire chorioid enormously expanded with blood, the clot in some places reaching the thickness of half an inch.

DETACHMENT OF THE CHORIOID.

This condition is not infrequently found in excised eyes, but clinically it is very rare indeed. The first cases were described by Von Graefe and Liebreich in 1854.

It may occur spontaneously, but more commonly it is caused by a blow, and a great many cases have occurred after extraction of cataract with loss of vitreous.

The impairment of vision comes on suddenly, and is accompanied by a defect in the field corresponding to the position of the detached chorioid. There is no pain, nor are there any external signs of inflammation. Tension is normal or reduced, and phthisis bulbi eventually takes place.

At some part of the fundus one sees, reaching into the vitreous and quite immovable, a uniform semi-spherical, slightly transparent elevation of an orange color, with irregular masses and mossy-like pieces of pigment on its surface, and behind the pigment one makes out a set of vessels which from their regularity and other characters are readily recognized as those of the chorioid. When the case is seen early and the characters of the affection are well marked, as in the above description, there may not be much difficulty in making a correct diagnosis, the absence of fluctuation or trembling differentiating it from simple detachment of the retina, and the visibility of the underlying chorioidal vessels distinguishing it from chorioidal sarcoma. Many cases, however, especially if seen at a later stage, are complicated by separation of the retina from the surface of both the detached and the undetached chorioid, which completely obscures the original condition. In other cases, as recorded by Michel and by Mules, the layer of chorioidal vessels becomes concealed, possibly by a layer of lymph.

Elschnig records a case in which detachment took place at the site of an old patch of chorioido-retinitis, and it is probable, as he suggests, that an inflammatory agglutination of the chorioid and retina is a usual antecedent,

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the detachment subsequently taking place from shrinking of the vitreous, as in the case of retinal detachment.

The greatest difficulty is often experienced in distinguishing detachment of the chorioid from sarcoma, and it is only by watching the case for some time and noticing the absence of further change, especially the absence of increase in the tension, that we are enabled to make the distinction.

In some instances, especially in detachment following cataract extraction, the prominence has spontaneously disappeared.

TUBERCULOSIS OF THE CHORIOID.

Tuberculosis of the chorioid is met with either as miliary tubercles in general acute tuberculosis or as large masses or infiltrations in chronic tuberculosis. The latter condition will be described under Tumors of the Chorioid.

Miliary Tubercles.—Miliary tubercles of the chorioid nearly always occur in both eyes. They vary in number from one to ten or more, but most frequently they average from three to six. When few in number they are confined to the neighborhood of the disk and the macula; when very numerous they reach to the equator.

They occur as round spots of a pale rose color, shading off gradually at their margins into the normal color of the chorioid without the presence of any pigment-ring. They are usually from one-third to two-thirds the diameter of the disk, but may be slightly larger than the disk. The larger ones are distinctly prominent, grayish yellow in color, and occasionally slightly pigmented at their margins.

The ophthalmoscopic appearances may be exactly like some of the most recent spots seen in ordinary disseminated chorioiditis, and this is not to be wondered at, as the latter affection is very often tubercular, being dependent upon a tuberculosis which is chronic, while tubercles in the chorioid occur in patients with acute general miliary tuberculosis and make their appearance only a few days or even hours before death, so that, practically considered, the two conditions could not possibly be confused. If the patients, however, were to live for any considerable time after the appearance of the chorioidal tubercles, it is, I think, probable that the changes in the fundus would ultimately come to resemble those described in cases of well-advanced chorioiditis disseminata.

The miliary tubercles, unlike the changes in chorioiditis disseminata, start in the deeper layers of the chorioid, growing mostly from the adventitia of the larger vessels, and anatomical examination in any case always reveals many more than could be seen before excision; for when confined to the deeper layers, or when very minute,—and they may exist as mere "dust,"—or when transparent, as they may be before retrogressive changes take place, their existence cannot be made out by the ophthalmoscope.

It is important to remember that they give rise to no defect of sight, and that repeated ophthalmoscopic examination is necessary to exclude

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their presence. Examination of the larger and older tubercles shows the usual microscopical characters of tubercle,—giant cells with a peripheral circle of nuclei surrounded by a reticulum of fibres, with small-cell infiltration, caseation, and sometimes extravasations of blood. The smaller tubercles appear simply as collections of lymphoid cells in spaces between the vessels. The overlying retina remains intact, save for its pigment-layer, which is broken through over some of the larger deposits.

Tubercle bacilli can by no means always be demonstrated. In six cases Lawford found them in two only, although in all they were easily found in the meninges of the brain; and many other observers have had a similar experience. Chorioidal tubercles are certainly common in general miliary tuberculosis. In eighteen cases of this disease, Cohnheim found them in every one; Demme found nineteen examples in eighty-nine cases of acute miliary tuberculosis in children; Angel Money found thirty-one per cent. on anatomical examination; in twenty-three globes excised from fifteen patients who died of this affection, Bock found them in nineteen. The higher estimates occur in anatomical and not in ophthalmoscopic observations.

They appear to have no definite relation to tubercular meningitis. Out of a large number of cases of this affection during twelve years' observations, Baxter met with no case of chorioidal tubercle. Sharkey found them only once in all the cases he had seen during three and a half years. Schreiber saw but one example in twenty cases of basilar meningitis.

Gowers, Cohnheim, Horner, and others think they are much rarer in tubercular meningitis than in general tuberculosis *without meningitis*, and the last-named says, "It seems as if the pia cerebri takes the place of the pia oculi." Barlow, however, in sixteen cases of tubercular meningitis found tubercles in the chorioid post mortem in thirteen, and, as tubercular meningitis is often only a part of general tuberculosis, he does not believe in the above-mentioned generalization.

Their detection by means of the ophthalmoscope may be of great service to the physician in enabling him to distinguish acute miliary tuberculosis from typhoid, which it sometimes resembles; but, of course, failure to detect them would not negative the former.

TUMORS OF THE CHORIOID.

From a clinical point of view there are only two sorts of tumor of practical importance, the tubercular and the sarcomatous, and the latter is by far the more frequent. We also meet with carcinoma, which is always metastatic, with cavernous angioma, with fibroma and fibro-chondroma, and with some few other rare varieties of growth.

Conglomerate Tubercle of the Chorioid.—In contrast to what has been found to be the case in miliary tubercle of the chorioid, conglomerate masses are met with in the subject of chronic tubercle whose general condition is not usually of such gravity as to overshadow the local affection; and, although the chorioidal disease is probably always secondary to tubercle

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elsewhere, we often find it difficult or impossible to prove the existence of the latter, and so have to arrive at a diagnosis partly from an examination of the condition of the eye and partly by a process of exclusion of other general diseases, especially syphilis.

The disease usually affects one eye only, and when, as very rarely happens, the second eye is attacked, this does not take place by direct extension from the first eye. Conglomerate tubercle of the chorioid may be characterized by the early appearance of destructive inflammation of the eyeball with perforation of its tunics, or it may occur as an ophthalmoscopic picture without external signs of inflammation, or a glaucomatous condition may in some few cases supervene.

I saw a good example of the first group in a boy, aged two years, whose left eye, lost after an inflammation of six weeks' duration, showed general conjunctival injection, opacity of the media, and a large scleral staphyloma at the outer and lower aspect. As seen in the specimen (Fig. 2), the posterior half of the staphyloma is occupied by a non-vascular gray exudation, breaking down in the centre, and apparently continuous with the sclerotic. The rest of the eye, with the anterior half of the staphyloma, is filled with stratified blood-clot, through which can be traced portions of the detached retina. Microscopic sections of the entire mass gave convincing proof of its tubercular nature,—numerous well-marked giant cells, with nuclei arranged round the periphery, surrounded by a fine net-work of fibrous tissue, caseating centres in abundance, and an absence of blood-vessels. No examination was made for bacilli.

The diagnosis of conglomerate tubercle of the chorioid was first made by Horner in a boy eight years of age who had inflammation of the eye for three months, with an ill-defined, flat, grayish tumor, which had perforated at the upper part of the eye, with thickening of, and nodules in, the conjunctiva of the bulb. The post-mortem examination confirmed the diagnosis, and showed direct communication of the intra- and extra-ocular portions of the growth.

Manz records a case in an eight-year-old boy who had an affection of the brain and eye and atrophy of both optic nerves, with detached retina and a grayish tumor beneath it in the left eye. Death took place five weeks later, and the tubercular nature of the growth was established.

These growths occasionally spring from the disk, as shown by Michel and others, and Brailey describes a case in which a tubercular mass grew from the disk and surrounding chorioid and somewhat resembled a glioma of the retina.

Some few years ago George Carpenter reported a number of cases in which large grayish masses of tubercle in the chorioid were recognized by the ophthalmoscope in ordinary chronic surgical tuberculosis; and there is no doubt that systematic ophthalmoscopic examination of such patients would show a larger proportion of conglomerate tubercle of the chorioid than is at present thought to exist.

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In the *Centralblatt für praktische Augenheilkunde* for November, 1888, Schöbl records a case in a woman aged thirty-six with a central blank in the field of the right eye and vision of 6/60. The optic disk was congested, and at its outer side was a very large area of retinal haze. A few months later this area was occupied by an uneven, pale tumor about one millimetre in height; the retina was diffusely pigmented, but not detached. Examination of the lungs gave a negative result. In a few months the eye became glaucomatous and the media opaque. The globe was excised. The patient died of pulmonary phthisis. Histological examination showed a tubercular growth of the chorioid in parts incorporated with the sclerotic. Bacilli were found.

I have had under observation for nearly three years a feeble, bronchitic old man with a large central chorioidal exudation in the right eye which I believe is tubercular. When first seen there was simply a small chorioidal hemorrhage, with some disturbance of the uveal pigment in its neighborhood. This was followed by the gradual development of a grayish, non-pigmented, rounded spot, which became slightly raised, and was crossed in front by the retinal vessels. Distinct cicatricial changes involving the retina and probably also the vitreous afterwards took place. I have another case, a single young woman, in whom the central exudation takes the form of a steep, white mound, with patches of pigment near the base, reminding one of a snow-capped mountain. Syphilis can be definitely excluded, and, although no unequivocal signs of phthisis are present, she has a chronic cough, and her father died from rapid pulmonary phthisis.

The ophthalmoscopic appearances are sometimes of a very diffuse nature, as in a case of Hirschberg's of a man aged twenty-seven years with tubercular meningitis, in whose left eye there was complete obscuration of the disk, with enormous distention and tortuosity of the veins, with hemorrhages and diffuse general white opacity of the eye-ground, due to wide-spread thickening of the chorioid, and infiltration by tubercular nodules, accompanied by widening of the surrounding vessels.

The difficulties in diagnosis vary according to the form in which the disease occurs.

When it takes the form of a general inflammation of the globe, with rapid formation of a staphyloma, it has to be distinguished from ordinary panophthalmitis and from specific disease. The former may be excluded by noting the absence of trauma or other of the conditions already mentioned under the causes of purulent chorioiditis, and by the painless course of the affection, while the latter always follows an acute gummatous iritis and is accompanied by other signs of syphilis.

From a practical point of view it is very important to remember that the capsule of the eye seems to soften and melt away before tubercular growths, staphyloma taking place rapidly without increase of tension, or with only very transitory increase; whereas in glioma, and especially in sarcoma, the sclerotic is very resistant, in the latter giving way only after a period of at least twelve months, or even several years, and then appar-

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ently more from the mechanical effect of the increasing tension than from a process of disorganization and softening.

When the conditions admit of ophthalmoscopic examination the disease appears as a solitary white or grayish-white, nodular, and always distinctly raised area under the retina, usually about the central region, and often accompanied by one or more minute satellite patches. It is readily distinguished from a white sarcoma of the chorioid or a retinal glioma by the absence of vessels and by the irregularity and want of definition of its borders; the disk, moreover, is often more or less blurred. It differs from a subretinal cysticercus by its immobility and its lack of translucency.

Even the ophthalmoscopic cases may afterwards become staphylomatous and perforate. When, as very rarely happens, well-marked glaucomatous symptoms appear, and the eye is seen for the first time in this condition with the media opaque, all we can infer is that there is an intra-ocular growth of some kind.

Prognosis and Treatment.—As the disease is probably always secondary, we are not justified in extirpating every bulb affected by tuberculosis, with the object of preventing general infection. This should be done only when the eye is lost, or when the case is of a markedly rapid and progressive character. In all other cases we may safely watch the eye and treat the case with cod-liver oil, creosote, and other constitutional remedies.

Death may take place, whether early enucleation has been done or not, from tuberculosis of the brain, the lungs, or other parts, but not, of course, with anything like the frequency that obtains in miliary tuberculosis of the chorioid, and very often not till after the lapse of many months or even of some years. I have recorded a death from tubercular meningitis nine months after early enucleation for a tubercular growth of the ciliary body. Manifestations of tubercle may show themselves for the first time at varying periods after removal of the eye.

From all this it appears more than probable that the dissemination of tubercle takes place, not from the affected eye, but from the lighting up into activity of some latent focus of disease in the interior of the body.

SARCOMA OF THE CHORIOID.

This is the chief intra-ocular growth of adult life, in contrast to glioma of the retina, which is mostly found in children of twelve months or less. It occurs within a much wider range of age than glioma,—from twenty years or even less to old age, usually between forty and fifty,—and is hence much more liable than glioma to be mistaken for other conditions.

General Characters.—Sarcoma of the chorioid is always primary, always single, and generally more or less deeply pigmented. It never affects both eyes. It arises by a broad base from the chorioid at any part of the fundus, and usually assumes the form of a single, rounded, knob-like process directed towards the centre of the eye, and separated from the base by a more or less distinct neck.

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Structure.—The free surface for most if not all of its extent will be found invested by the vitreous lamina and uveal pigment, and even the chorio-capillaries can often be traced. The "neck" sometimes, but by no means always, corresponds to where the vitreous lamina has been broken through.

The growth is almost entirely composed of pigment-containing cells, with an insignificant quantity of intercellular substance. The blood-vessels are thin-walled, and extravasations of blood are frequently present.

The pigment is, as a rule, more abundant near the sclerotic, but it varies in different parts of the same growth, being almost if not entirely absent in some places and very abundant in others.

The cells are mostly long or short "spindles," with a single nucleus in the centre, the ends tapering off into fine filaments; they are often arranged in a whorled manner round the vessels, mapping out the growth into rounded areas with a cross-section of a vessel in the centre of each. The cells next the vessels stain more deeply than those at the periphery. Round cells are also met with, and the growth may exceptionally be chiefly or entirely composed of these.

Frequency.—As regards the frequency of its occurrence, I may state that in the period of eight and a half years from January 1, 1880, to June 30, 1888, we had seen forty cases among a total of one hundred and nineteen thousand five hundred eye-patients, giving a percentage of .03, which may be compared with Freudenthal's .04 (twenty-four cases), Hirschberg's .05 (thirteen cases), and Fuchs's .06 (ninety-one cases from the clinics of ten different ophthalmic surgeons in Germany). The disease is said by most observers to occur with about equal frequency in the two sexes, but in the forty cases already referred to we found it twenty-five per cent. commoner in males, and in Lawford's one hundred and three cases it was fifteen per cent. commoner.

Causation.—Nothing is known as to causation. Blows or other injuries have nothing to do with it, nor has the occurrence of tumors in other members of the family. The persons affected are quite up to the average in point of health.

Starting-point.—The growth usually starts in the larger vessels, and extension, when it occurs, takes place by the vascular channels. There is seldom, if ever, any implication of the preparotid or other lymph glands. Fuchs demonstrated the presence of sarcoma cells within the capillaries of the chorio-capillaris, and Landesberg showed microscopical tumor accumulations between the chorio-capillaris and the lamina in an eye removed from an eight-year-old boy with a spindle-celled sarcoma of the ciliary body.

Stages.—It occurs in three stages. In the early period of growth the tension is normal or subnormal, and the eye is free from inflammation or opacity of the media. This stage usually lasts from six to twelve months, but may exceptionally extend much longer, even six years, as in a case recorded by Hirschberg.

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The second stage is characterized by increased tension, injection of the bulb with opacity of the media soon following, and lastly the growth enters upon the third stage by bursting through the capsule of the eye, as evidenced by sudden reduction of tension and, if the site of the rupture be not too far back, the appearance of a dark mass outside.

Diagnosis.—The chief point we shall have to decide in any case is, Are we dealing with an intra-ocular growth or some condition simulating this? The particular variety other than glioma or sarcoma cannot be diagnosed clinically, and these differ so widely in appearance and especially in the age at which they occur that they cannot be mistaken one for the other.

As a rule, the patient does not become aware of the defect in his sight until the tumor has attained a considerable size, when central vision is abolished and the defect in the field is absolute and sharply defined.

Paralysis of a segment of the iris, and fulness of the anterior veins of the globe corresponding to the site of the growth, are often noticeable, and enable us to diagnose the condition before using the ophthalmoscope. The first mentioned of these signs is due to implication of the ciliary nerves in the growth, and the fulness of the anterior veins is due to the tumor preventing the escape of the blood by the usual outlets, the vasa vorticosa.

When sarcoma occurs at the posterior pole of the eye or near the ciliary region, the growth is closely invested by the retina, no serum intervening, and direct inspection of the tumor with its vessels renders the diagnosis unmistakable.

The growth is always best seen by the mirror alone, or, if situated close behind the lens, it appears by oblique illumination as a rounded opaque prominence, varying in different cases from a lightish yellow tint to a brown or a deep black. The retinal vessels can be traced over its surface, and often one can make out a deeper mesh-work of vessels belonging to the growth itself.

Serum.—In all other situations than the posterior pole or ciliary region there is almost always the presence of more or less serum between the growth and the overlying retina, a condition which may render a positive diagnosis very difficult or impossible. In fact, this constitutes by far the commonest obstacle to a direct diagnosis in the first stage, as it is liable to be mistaken for simple detachment of the retina.

The serous effusion has by some been looked upon as an exudation from the surface of the tumor itself, but it is generally regarded, and I think rightly, as caused by the pressure on the large emergent veins of the chorioid, which also explains its absence when the growth arises at the posterior pole of the eye or far forward, in both of which positions, as we know, there are no emergent vessels; and, further, the ciliary part of the retina is strongly adherent to the uveal layer, while elsewhere the attachment is but slight.

This serous effusion is certainly the rule, and is by some regarded as a constant and invariable accompaniment of chorioidal sarcoma. I think,

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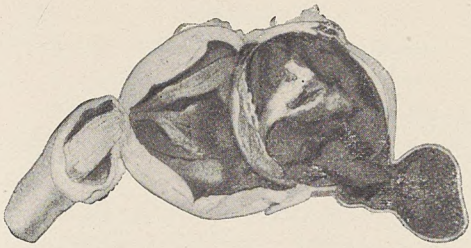
FIG. 5.

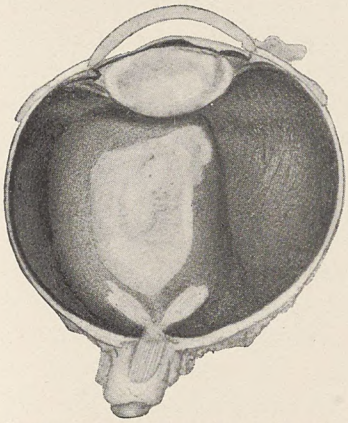


Melanotic sarcoma of seventeen years' duration, showing shrinking and perforation of globe, and the presence of a bony spicule.

FIG. 6.

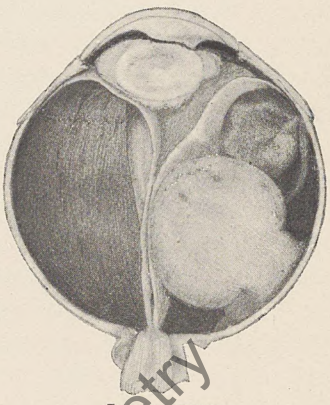
Unusual cake-like form of chorioidal sarcoma with overlying retina quite adherent.

FIG. 7.



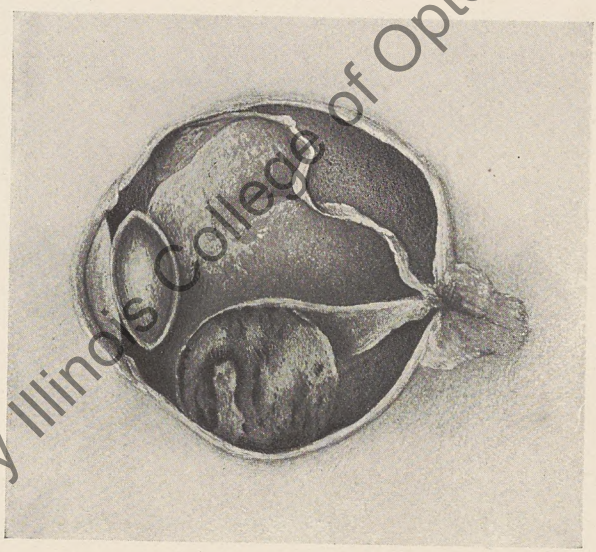
Leuco-sarcoma: free surface entirely invaded by hexagonal pigment layer.

FIG. 8.



Leuco-sarcoma.

FIG. 9.



Melanotic sarcoma with bulging of sclerotic corresponding to site of tumor.

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however, that this is true only of growths occurring about the equator, and when they have attained a considerable size. I have certainly seen several central sarcomas at an early stage in which there was an entire absence of subretinal effusion. As for tumors about the equator, I cannot say, as they are never seen by us at an early stage.

Retina adherent to Tumor.—I have seen several examples of large intra-ocular sarcoma growing from the equatorial regions, in which there was a most intimate union between the growth and the overlying retina, probably due to adhesive inflammation, the attachment being so complete that even in microscopic sections it was not freed. (See Figs. 6 and 9.)

In such cases the diagnosis would not be very difficult, even although the retina were elsewhere extensively detached.

Simulating Simple Detachment of the Retina.—In any case of suspicious detachment of the retina,—and by suspicious I mean occurring in one eye only,—in the absence of high myopia, a severe blow on the eye, or a wound of the sclerotic, and especially if occurring in an adult, one must bear in mind that a chorioidal sarcoma may be present, and examine, by means of the ophthalmoscope, very carefully from time to time, with a well-dilated pupil and the strongest possible illumination. If there be only a small amount of serum under the portion of retina covering the tumor, the usual knob-like character may be well marked, and the diagnosis may be in consequence easy; this is especially likely to be the case if the tumor arise in the upper segment of the eye, for the subretinal effusion will gravitate to the lower part, and leave the tumor open to our inspection.

It must, however, be confessed that in some cases, if the retinal detachment be large, it may be impossible to give a positive opinion. I select the following, as it affords a good example of the difficulty we are discussing, and is of interest in other respects.

With Rupture of Retina.—Case: Job Atkins (Dr. Glascott), aged thirty-one, painter. When first seen had lost the sight of the left eye for eighteen days; at least he found out the defect then. There was no history of injury to the eye, and the sight was perfect in the other eye. We found in the affected eye a large retinal detachment at the inner and lower part, there was increase of tension, and all perception of sight was lost. The patient had several times suffered from lead colic and rheumatism in the shoulders and feet, but his general state of health was good. Urine and heart normal.

With Amblyopia of the Other Eye.—At the second visit, one week later, the vision of the good eye had sunk to 16 Jäger, but the fundus was absolutely normal. The field of vision for white, red, and green was normal, and there was no central scotoma for color. On examination three months later it was found that the detached retina had ruptured, the ragged edges of the tear being plainly visible, and disclosing a black, knob-like growth at the nasal side, just behind the lens.

The eye was enucleated shortly after this, and the pathological examination confirmed the diagnosis of melanotic sarcoma of the chorioid. This

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is the only occasion in which I have known rupture of the retina to take place and the presence of the tumor to be thus made manifest.

The vision of the right eye commenced to improve soon after enucleation, reaching 10 Jäger and 6/36 in one month, 4 and 6/36 three months later, and 1 and 6/6 shortly afterwards. I am unable to say what was the nature of this transient form of amblyopia, but I am strongly inclined to regard it as dependent in some way on the presence of the growth in the other eye. The sight was perfect seven years later, and there had been no attack of dimness of sight. I have seen and recorded a case of retinal glioma in which, after enucleation, the sound eye went quite blind without ophthalmoscopic change.

Increase of Tension with Retinal Detachment.—When increase of tension occurs in a case of retinal detachment, even if the eye be not in a state of acute glaucoma and the globe be still free from general injection, the case is no longer one merely of suspicious detachment, but is almost certainly one of chorioidal sarcoma. It would be easy to multiply cases in which chorioidal sarcoma has been correctly diagnosed on this evidence alone, but the following will suffice.

Esther Breakell (Dr. Little), aged twenty. Sight in the left eye gone for three months; defective for nine months, with attacks of dimness.

The retina was totally detached and crowded against the back of the clear lens; it did not move about on movements of the eye. The tension was very high, and there was no translucency.

On excision we found a melanotic sarcoma the size of a filbert arising from the posterior segment of the eye, surrounding the disk for some distance on every side, and enclosing in its substance the retina, which could easily be traced from behind forward.

Increase of Tension and Retinal Detachment without Growth.—As above stated, the occurrence of increased tension in retinal detachment is alone sufficient evidence of intra-ocular growth; but if there be also inflammation with posterior synechiæ, and especially if the increase of tension comes on only after the use of atropine, as in the following case, there may be no tumor.

Elizabeth Walmsley, aged thirty-five. Gave a history of gradual failure of vision in the right eye for eight months, commencing on the third day after her confinement. There had been no pain till the day before she came under my care.

We found faint injection of the eye. The pupil was bound down to the lens-capsule by numerous adhesions at the lower part; the iris was slightly discolored; there was but slight illumination at the outer side, not sufficient to enable one to make out any details in the fundus. At the nasal side I thought I could detect some detachment of the retina far forward. The tension was normal.

I regarded the case as one of those forms of septic inflammation—in this instance an irido-cyclitis—which sometimes follow confinement, and

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ordered iodide of potassium internally, and atropine to be applied to the eye.

One month later we found the tension increased to a high degree, and just behind the lens at the nasal side one could see a rounded, buff-colored surface with vessels coursing over it. I felt compelled to change my opinion of the nature of the case, for the presence of retinal detachment, increase of tension, and vascular mass pointed with irresistible force to sarcoma of the chorioid, and, further, the loss of sight, preceding for a period of several months the occurrence of pain and injection of the eye, was quite in accordance with this view of the case.

On section of the excised bulb we did not find a tumor, as we anticipated, but only detachment of the retina, a fold of which was adherent to the posterior surface of the lens, and had given rise to the appearance of a solid growth.

Chronic Localized Chorioiditis with Deposit of Bone.—Detachment of retina with increased tension may also be caused by a chronic localized chorioiditis, as in the following case. Jane Wilson, aged fourteen years and eleven months (Dr. Little), had lost the sight of the left eye certainly for six months, and probably it had been failing for eighteen months. There was no perception of light, T = +3; the cornea was hazy, the pupil dilated *ad maximum*, only a very narrow rim of iris to be seen covered with pigment on its anterior surface. There was total separation of the retina, which formed a funnel-shaped cavity extending from the disk to the ciliary attachment. A dirty-yellowish reflex was obtained from the interior of the eye. The detached retina was thickened and discolored, and did not wave about; splashes of blood could be seen on it in places.

During the few days the case was under observation the haziness of the media increased, and, the diagnosis of intra-ocular growth having been arrived at, the eye was removed.

On section of the globe we found the detached retina much thickened, especially at its posterior attachment, where it forms a pinkish-gray, fusiform swelling, whose base is closely adherent to the chorioid at the temporal side of the disk. The chorioid is at this part much thickened and engorged with blood, and shows embedded in its substance several plates of young bone. Its structure is here quite continuous with the fusiform swelling formed by the detached retina.

Strangely enough, since writing the above I have had to remove an eye from this patient's elder sister, aged twenty years, for a similar condition.

Cases evidently of a similar nature, and giving rise to similar symptoms, have been recorded by Lawford and Treacher Collins, the former under the title of "New Tissue-Formation on Inner Surface of Choroid;" the latter, "Neoplasm between Choroid and Retina." It appears that these fibro-plastic formations start in the superficial layers of the chorioid and burst through the vitreous lamina.

Simulating Acute Glaucoma.—Chorioidal sarcoma often comes under

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our notice with all the appearances of an acute inflammatory glaucoma,—that is, with increased tension, total or nearly total loss of sight, severe shooting pains, general injection of the eye, a dilated and fixed pupil, haziness of the cornea, a shallow anterior chamber, and opacity of the media. It is of the first importance in this the second stage of sarcoma to make an accurate diagnosis, as iridectomy is just as strongly indicated in glaucoma as it is contra-indicated in chorioidal sarcoma,—in the former condition curing the disease and, if done in time, restoring the sight permanently, while in the latter condition such a procedure could only do harm by delaying the enucleation and in all probability hastening the fatal issue by giving rise to infection of the neighboring tissues.

On closely questioning the patient we will often elicit the information, when the glaucoma is secondary to intra-ocular growth, that the sight has been lost for some considerable period before the onset of inflammation, and, further, that the premonitory symptoms usual in primary glaucoma—*i.e.*, transient attacks of pain, dimness of vision, and colored rings, coming on at night and disappearing after a sleep—have not been observed. Another very important point in the differential diagnosis is that there are no remissions in the symptoms, such as we find in primary glaucoma.

When a glaucomatous eye is protruded and slightly fixed, this is strongly in favor of chorioidal sarcoma, and is due to inflammatory masses adherent to the external surface of the globe.

If all perception of sight be gone and the eye be painful, we would enucleate without regard to the diagnosis, as iridectomy is never of any use in glaucoma which has reached this stage; but if some little sight remains it is very desirable to make a diagnosis, and I have found the estimation of the visual field by the candle of some service, the retention of the nasal side being against glaucoma, in which disease, as we know, this portion of the field is always the first to go. In spite of all care, however, in the history and objective examination, sarcoma has frequently been found in lost eyes enucleated for glaucoma, and iridectomy has been done in eyes containing sarcoma: so it is certain that this mistake cannot be altogether avoided.

Intra-Ocular Hemorrhage simulating a Growth.—Retinal hemorrhage is sometimes quickly followed by glaucoma, and if this occurs with a single large blood-clot it might lead us to an erroneous diagnosis. This occurred with me in the case of an adult who had lost the sight of one eye. There was increased tension and a large, dark, dense, opaque, prominent mass in the lower part of the fundus; hand-movements could be recognized only in the lower part of the field. Excision revealed simply a very dark blood-clot, several millimetres in thickness, lying on and closely adherent to the lower part of the retina. Webster records a similar experience.

Bulgings of the Sclerotic.—Localized bulgings of the sclerotic occasionally take place in chorioidal sarcoma, and, in fact, are the rule in sarcoma at the posterior pole of the eye, as in a case of mine published in the

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Archives of Ophthalmology, vol. xvii. part 2. Here, as in one or two others of my specimens of sarcoma at other parts of the chorioid, the bulging corresponds with the site of the growth; but this is not always the case, as I shall show, and, further, such bulgings occur perhaps more frequently from other conditions than intra-ocular sarcoma, as in the following:

James Mackie (Dr. Glascott), aged nineteen. Left eye blind for four years; no accident; no pain or inflammation at any time. There was a large ciliary staphyloma upward and outward, which had been noticed by the patient for a fortnight. There was only slight local injection; no pain or tenderness on pressure; the tension was very high; the lens was partially opaque, preventing an ophthalmoscopic examination. On enucleation no tumor was found, but only the staphyloma formed by the expansion of the sclerotic and ciliary portion of the uveal tract.

I should here mention that Dr. Mules suggested throwing a strong light on the outer surface of the bulging, to see if the pupil could thus be illuminated from behind, and he satisfied himself from this test that there was no solid growth. This is no doubt a test worth remembering, and is very valuable as evidence of the presence or absence of a tumor situated at the thinned portion of the sclerotic (when palpation with the fingers might also be of service); but where, as in the next case, the tumor did not correspond with the staphyloma, it would not be safe to negative the existence of a growth by this test.

Mary Gradwell (Dr. Little), aged thirty-three. Vision bad in left eye three months; read No. 19 Jäger. There was a large separation of the retina *below*, T = —1. Patient returned in two months with T = + and a ciliary staphyloma *above*. On enucleation, the lower half of the eye was found to be occupied by a melanotic sarcoma, and the staphyloma above was quite free from the growth. There was complete detachment of the retina. (Fig. 3.) The rapidly forming staphyloma in tuberculosis has been already referred to.

Rupture of the Capsule of the Eye.—Fig. 4 is a fine example of melanotic sarcoma which has reached the third stage, the portion of growth outside the sclerotic being nearly as large as that within the eye. The patient was a woman aged fifty, and at the date of the enucleation the tension was normal, the lens was opaque and calcareous, there was no anterior chamber, and the iris was adherent at several points to the lens-capsule. She had been seen by a competent observer six years before, who had diagnosed the condition as "old irido-chorioiditis with ciliary staphyloma."

The great power which the sclerotic possesses of resisting for years the action of sarcoma has an interesting parallel in the case of another fibrous structure, the large blood-vessels of the limbs, which may often be seen, on the operating-table, running uninjured through a huge sarcomatous growth which may have extensively involved the other tissues.

The growth may make its escape through the cornea when this structure, as sometimes happens, undergoes ulceration.

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Once the capsule of the eye has given way, the rate of growth in the tumor becomes much more rapid, and a large fungating mass is formed.

Episcleral Nodules.—When episcleral nodules are present, they occur near the entrance of the optic nerve, or at the equator, the path of exit being along the track of a perforating blood-vessel; but, as these nodules do not occur farther forward, their presence is recognized only on the excision of the globe: hence they cannot be used as aids to diagnosis.

Protracted Cases.—The course of sarcoma is sometimes very protracted, as is well illustrated in the following:

Jeremiah Turner (Dr. Glascott), aged forty-eight, stated, without being questioned, that he had been seen at this hospital seventeen years ago, and was then told by Mr. Windsor that he had a cancer at the back of the eye, which ought to be removed. He declined operation, however, and the eye kept free from pain until quite recently, when a small black knob appeared through an aperture in the anterior part of the shrunken globe. Fig. 5 represents an antero-posterior section of the eye, and shows a melanotic sarcoma with a spicule of bone in its interior, by no means a common occurrence in this condition, but not nearly so rare as the formation of true cartilage, examples of which have been recorded by Knapp and Alt.

Shrunken Globes.—Von Graefe long ago pointed out that phthisical bulbs sometimes contained sarcomatous growths, and that such shrunken globes, although often the site of severe spontaneous pain, were free from tenderness, shortened in their antero-posterior diameter, and often pushed out of the orbit by a sarcomatous mass behind the eye.

One frequently reads accounts of sarcoma occurring in lost and shrunken eyes, which are hence supposed to be especially liable to this affection, but I feel pretty sure that these have been really examples of sarcoma causing phthisis bulbi. Such a case, with the correct explanation, is recorded by Guttmann, in which the growth had been present for thirty years.

Irido-Dialysis.—Separation of the iris at its ciliary attachment often occurs in sarcoma of the ciliary body from direct implication of the base of the iris, and is of great diagnostic importance in this condition. I am not aware that it ever takes place in sarcoma confined to the chorioid proper, even when the tension is very high, nor do I recall its occurrence in primary glaucoma: hence I believe that simple increase of tension has no place in its production; and, further, the tension has been normal or subnormal in the cases I have seen.

Simulating Chronic Iritis.—In very exceptional cases a sarcoma may come before us with the appearances of a simple chronic form of iritis or irido-cyclitis, with closure of the pupil and opacity of the media, unattended by increase of tension, pain, or injection of the eye. The following case, which I published in the *Ophthalmic Review*, December, 1891, is the only example that has come under my observation.

Male, aged sixty, consulted me on account of loss of sight in the left eye, which had been failing for about eight months, and was unattended

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by pain or inflammation. There was no perception of light; the tension was normal; there was just a trace of injection of the eye; the pupil was of moderate size, but bound down to the lens-capsule by several adhesions; the iris was somewhat degenerated, reddish brown in color, and bulging in places; the cornea and apparently the lens were transparent, but no reflex whatever could be obtained by the ophthalmoscope.

I ordered him to use atropine and see me again shortly, hoping that I should then be able to illuminate the eye and arrive at a diagnosis. I was called to see him three days later, and found the eye of stony hardness, much injected, and excessively painful. The pupil was very slightly dilated, and, as before, no reflex was obtainable. The atropine was at once stopped and eserine substituted. A hypodermic injection of morphine was given each night, and iced compresses, and afterwards hot fomentations, were used.

In spite of all these measures, the eye remained glaucomatous and the pain did not in the least abate.

On thinking over the features of the case, it appeared to me untenable that a chronic and painless iritis could utterly destroy the sight without any shrinking or even diminution of tension of the globe; and on closely questioning the patient, it was ascertained that the dulness of sight began as an opaque black shade from *above* (not from the nasal side, as in glaucoma), which kept gradually extending in a downward direction, and I felt convinced from this that I had to deal with an intra-ocular growth.

On section of the excised eye, we found that it was more than half filled with an intensely melanotic sarcoma of the choriod.

In such cases as the above the use of atropine has been recommended for the purpose of diagnosis, the supervention of the glaucomatous stage showing the presence of a growth; but I must say that I had no such idea in view when I ordered this agent, and, further, I do not think this test is reliable when iritic adhesions are present, as is shown in the case of Elizabeth Walmsley (p. 368).

Early Stage.—In sarcoma at the equator or near the ciliary region no complaint, as a rule, is made till the tumor has attained a considerable size, and in the large majority of cases, when seen by the surgeon, only one opinion can be entertained as to the nature of the ophthalmoscopic changes. On the other hand, in sarcoma commencing at the macula the patient is very soon aware of something wrong with the sight, and for this reason we shall be called upon for a diagnosis at a very early stage of the growth, when observers of great experience and recognized skill may fail to make out the true condition, as in cases recorded by Bowman, Hutchinson, Lawson, and Otto Becker.

I had a case of this kind under my care some years ago and utterly failed to diagnose it.

Samuel Done, aged thirty-nine, complained of dimness of sight in the right eye for three weeks. The vision was 6/36. A little above and to

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the inner side of the entrance of the optic nerve was a circular grayish-white patch about the size of the disk, over which could be seen coursing the retinal vessels. Its surface was level with the chorioid and showed a few patches of pigment; its edge was sharply defined.

With the exception of some slight pigmentary disturbance in the immediate neighborhood of the patch, there were no other ophthalmoscopic changes.

The diagnosis arrived at was simply exudative chorioido-retinitis. The case was examined from time to time for a period of one or two months, but no further changes took place, except that on one occasion I noticed that the patch seemed slightly raised.

Six months after the first visit, and a considerable period subsequent to my last ophthalmoscopic examination, we found that an enormous separation of the retina had taken place, the detachment reaching right up to the disk all round, and being easily seen by oblique illumination. Fingers could be counted with difficulty, and only at the centre of the field; the tension was lowered.

The eye was excised three months later for pain and increased tension, and was found to contain an enormous chorioidal sarcoma which filled the posterior half of the globe and was for the most part moderately pigmented, but close to the sclerotic behind and for a little distance forward it had a gray color.

This case shows as well as a single example can that the early stage of chorioidal sarcoma is not accompanied by detachment of the retina.

The diagnosis of the affection in its earliest stage possesses a peculiar fascination, and a good many cases have been recorded in which the eye has been extirpated when the vision was only slightly lowered and the growth itself no bigger than a millet-seed. It must be said that in the recorded cases pathological examination has invariably confirmed the presence of a growth, but we have no means of ascertaining how often this has not been the case and in consequence a useful eye has been sacrificed. Kniess enucleated an eye when the vision was equal to No. 8 Jäger, the growth being only the size of a millet-seed. When seen for the first time, twelve months before, the vision was normal on correction of .75 D. of myopia. There was very strong injection of the retinal vessels at the macula, but no other ophthalmoscopic change. The refraction subsequently became hypermetropic as the tumor developed at the macula. The presence at the macula of a single, sharply defined, round, opaque, dark gray spot, as if a wafer had been stuck upon the fundus, with its surface raised and traversed by the retinal vessels, is much more likely to be due to a chorioidal sarcoma than to any other condition. Before advising removal of the eye, we ought to convince ourselves that the spot is increasing in size by repeated examination of the fundus and measurement of the scotoma in the field of vision. It is far better to watch the case for a few weeks, or even a few months, than to make so serious a mistake as to enucleate a useful eye.

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I have certainly seen a few cases of central chorioidal changes, notably one of congenital pigment-spot, very closely simulating sarcoma, which proved not to be of this nature.

Unusual Shape.—The following case is recorded on account of the very unusual shape of the growth. It could not be seen ophthalmoscopically, and if it had been, I think it very likely that the diagnosis might not have been correct.

Betsy Fletcher (Dr. Little), forty-one years of age. The left eye had been defective for nearly two years; painful and bloodshot for about four months. The episcleral vessels were injected, the anterior chamber shallow, the pupil semi-dilated and motionless, the media hazy, the fundus invisible, and the tension increased.

Fig. 6 represents the upper half of the eye, with a flat, cake-like, pigmented sarcoma reaching from the disk to the anterior part of the chorioid at the temporal side. It is only slightly raised above the level of the surrounding chorioid. The free surface shows several rounded elevations, and has the retina so closely applied and adherent that even in microscopic sections there was no separation.

I have not seen another example of chorioidal sarcoma with this unusual shape; but it appears that carcinoma, which is exceedingly rare, and, unlike sarcoma, is always secondary, takes this cake-like or shell-like form.

Leuco-Sarcoma.—Non-pigmented sarcoma is said to be more frequent in the younger patients, and in the middle or anterior portions of the chorioid. We meet with one for every ten or fifteen of the pigmented variety. I have myself seen only two well-marked specimens, the cut surface in one of these being almost as white as paper. The patient, a man aged thirty-two, came under my care with a large yellowish-brown tumor at the upper part of the fundus of the left eye. The vision was reduced to fingers; the tension was slightly diminished.

The pigment-layer of the retina invested the entire free surface of the growth, so that it was only on slicing off a piece of the tumor that we became aware it was a leuco-sarcoma. (See Fig. 7.)

The other specimen, of which Fig. 8 is a representation, was from a patient of Dr. Little's whom I was afforded an opportunity of seeing, and, as in the other case, the ophthalmoscopic appearance was by no means so striking as the pathological preparation.

I should imagine that an ophthalmoscopic diagnosis of leuco-sarcoma in contradistinction to melanotic sarcoma could be made only either when the pigment of the hexagonal pigment-layer was absent, as in the albino, or where this layer had been broken through by the growth. (Fig. 9.)

Treatment and Diagnosis.—Nothing short of extirpation of the eyeball is of any use in chorioidal sarcoma, and this ought to be done as soon as one can make a positive diagnosis.

The optic nerve should be cut as far back as possible. I find from

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measurements that one may often cut it from fourteen to sixteen millimetres long. If after excision of the eye the nerve is found to have been cut too short, one should endeavor to pick up and excise as long a piece as possible, —by no means an easy proceeding.

There are two dangers which may arise after removal of the eye. The one is "local recurrence" of the growth in the orbital tissues, requiring a secondary operation and not necessarily fatal, and the other "metastasis" to distant organs, always speedily fatal.

As Fuchs points out, local recurrence means that some bud of the process has been left in the orbit, while metastasis implies the presence in the circulation of tumor-cells.

The former much rarer condition—about eight per cent.—may practically be altogether prevented by early enucleation, while the latter, which constitutes the chief source of danger, may start at any period of the primary growth.

The two processes are quite distinct. A patient may die of exhaustion from local recurrence without a trace of metastasis being found on post-mortem examination, and, as is well known, the converse of this is much more common.

If local recurrence takes place, it is usually recognizable within a very few months of the enucleation, although in some rare instances not for a much longer period.

Under these circumstances it is always advisable to clear out the entire contents of the orbit, which is best done in the following manner. The lids, having been disunited by free section at the outer canthus, are kept separated as widely as possible, and an incision is then made all round the margin of the orbit, within the lids, down to the bone. The orbital periosteum is detached as completely as possible by means of a raspatory, after which the orbital contents are seized and drawn forward by means of a strong pair of tenaculum forceps, divided close to the apex of the orbit, and removed. After arrest of the hemorrhage the cavity is stuffed with iodoform gauze. This proceeding is often followed by a permanent cure, for, fortunately, it is very rare for the recurrent nodules to spread to the bony walls of the orbit or to make their way into the cranial cavity. Pflüger removed a melanotic sarcoma which had already attacked the whole orbit, the antrum of Highmore, and the intra-cranial part of the nerve. After death, six months later, there was found intra-cranial sarcoma at the base of the skull, with metastases in the skin, muscles, and serous membranes.

Infiltration of the orbit is to be feared only when the eye has been glaucomatous for some time, more especially when perforation has already taken place, and in the latter condition enucleation should always be combined with removal of the orbital contents.

As before mentioned, recurrence in the orbit practically never takes place when enucleation is done in the first stage, but it may exceptionally

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occur before perforation can be made out by the naked eye, and in these cases microscopic examination shows infiltration of the sclerotic with pigment.

Metastasis to internal organs, by far the chief cause of death, may take place at any stage. It occurs almost invariably within two years of the date of operation, and in estimating the proportion of cases we include only such as are well at least three years after removal of the eye.

Von Graefe, writing many years ago, said that he had never seen a case well more than four years after operation, and Fuchs in two hundred and forty-three collected cases found only six per cent. alive after four years. I feel sure that some source of error has crept in here; at any rate, no other observer estimates the cures at so low a rate.

Among twenty-three enucleations for chorioidal sarcoma, I found that fourteen, or about sixty per cent., were alive and well from three to ten years after operation, a proportion of cures which agrees very closely with Martin's sixty-two per cent. in thirty-five cases and Lawford and Collins's fifty per cent. in seventy-nine cases, though considerably higher than Freudenthal's thirty-seven per cent. in twenty-four cases and Fuchs's twenty-three per cent. in seventeen cases.

From my own experience, I should say that fifty per cent. may be reckoned as permanent cures.

I can confirm Fuchs's interesting observation that the stage at which the eye is enucleated has little if anything to do with the occurrence of metastasis. For example, one of my cases, in which the eye was enucleated in the third stage, and when the growth had already been present for seventeen years, recovered, while two cases, in which the operation was performed at a very early period, with vision of 6/36 and 16 Jäger respectively, died.

I do not feel justified in drawing any deduction as regards prognosis from the histological characters of the growths in my own cases, although I accept as probably true the oft-repeated dictum of the microscopists that the round-celled and highly vascular growths are more malignant than the spindle-celled and less vascular ones. An examination of the ages of the patients in my own cases seems to point very decidedly to a better prognosis for the youthful than for those of more advanced years. The average age of the recoveries is 38.3, of the fatal cases 52.1, showing a difference of 13.8, and, what is perhaps still more striking, the four unusually young patients, aged respectively seventeen, twenty, twenty, and twenty-two, are all to be found in the list of recoveries.

The subject appears to have received very little attention at the hands of English ophthalmic surgeons, and in the text-books, with the exception of Berry's, where it is gone into very thoroughly, I do not find any reference to it.

Physicians, on the other hand, have not been slow to recognize intra-ocular sarcoma as the source of disseminated disease of a similar nature in

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other parts of the body. Murchison, in the second edition of his work on Diseases of the Liver, 1877, mentions a case of spindle-celled sarcoma of the liver in a man aged thirty who suffered from pain in the right side for eighteen months, with enlargement of the liver for one month. The left eyeball had been excised by Hulke for malignant tumor nine years before, the sight having been failing for two years. The patient died in twelve month from sarcoma of the liver, which weighed twenty pounds. There was no vomiting, ascites, or jaundice. Wickham Legg (*Lancet*, 1883, vol. ii. p. 1128) read at the Pathological Society of London the case of a man aged fifty-seven whose left eyeball had been removed by Streatfeild for melanotic sarcoma. Six months before death, darkening of the skin took place. This was uniform and not in patches, was most marked on the face and neck, less so on the hands, and not so obvious on the rest of the body. Norman Moore (*The Lancet*, 1889, vol. i. p. 577) showed at the Pathological Society of London a sarcomatous liver weighing sixteen pounds, which occurred in a man aged forty-eight who had been ill four months and whose right eye had been removed for melanotic sarcoma three years before. At the same meeting Sir William Lawrence mentioned two fatal cases after removal of eyes containing growths. Saundby (*Birmingham Medical Journal*, 1891, p. 147) mentions a case of widely spread melanotic sarcoma, with melanuria, in a woman aged forty-one, whose left eyeball, shrunken from an old injury received in childhood, contained a melanotic sarcoma which had burst through the sclerotic and formed a tumor outside the size of a bantam's egg. The author considered this to have been the starting-point of the affection.

The secondary growths are usually but by no means always pigmented, even when the primary growth is of this character. They occur generally in the liver and stomach, but often also in the lungs, heart, kidneys, intestinal mucous membrane, skin, serous membranes, and muscles; very rarely in the cranial and spinal cavities.

At the post-mortem examination of one of my cases we found the sarcomatous mass limited to the spinal cavity, filling up the space around the cord. During life the patient had occasional weakness of the lower limbs, with pain in the back, relieved by constantly lying on his face with a pillow under his belly.

Krause found black molecules in the vomit and urine. Von Jaksch's test for melanin in the urine, which is turned black by the addition of perchloride of iron, might be useful in giving a prognosis.

Before leaving the subject of chorioidal growths we shall make some remarks on some forms of tumor other than sarcoma; but, as these are very rare, present no distinctive ophthalmoscopic appearances, and are chiefly of pathological interest, a brief notice only is required.

Of late years there have been recorded some fifteen cases of carcinoma which differ markedly from sarcoma in the following particulars: they often affect *both* eyes, one shortly after the other; they are always *secondary*,

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the primary growth being in the large majority in the mamma, very rarely in the lung or the liver; they are lightish yellow in color and shell-like in shape, spreading far forward; pain is usually present, and death always takes place rapidly from widely spread metastases.

Jennings Mills, Lawford, F. Giulini, Schiess-Gemuseus, and Nordenson record cases of cavernous angioma of the chorioid. This form of growth occurs in young people, and is usually associated with congenital nævus of the face; the tumors have been recognized only on excision of lost and painful eyes with increased tension. In Giulini's case the growth was composed of rounded, elongated, and variously shaped spaces of all sizes, closely packed with red blood-corpuscles, very few white ones; the spaces were lined with endothelium, and separated by very fine connective-tissue strands. The tumor originated in that part of the chorioid adjacent to the supra-chorioid.

Plexiform angio-fibroma and enchondroma have also been met with.

DISEASES OF THE VITREOUS.

The vitreous is a colorless, transparent, gelatinous body which fills the posterior cavity of the eye. It is completely enveloped by a delicate transparent structure called the *hyaloid membrane;* its anterior surface is hollowed out to receive the crystalline lens, and on its other aspects it is in immediate contact with the ciliary body, the retina, and the optic disk.

The nutrition of the vitreous is dependent upon the vessels of the ciliary body, and to a lesser extent upon those of the retina, but no vessels enter its substance. It is traversed from behind forward by the *hyaloid canal*, a perfectly transparent tube two millimetres in diameter, extending from the optic disk to the posterior surface of the lens, which during fœtal life encloses the hyaloid artery, and afterwards serves as a lymph-channel.

Forty years ago Hannover and Brücke showed that the vitreous body consists of a semi-fluid portion enclosed in the meshes of a transparent framework of delicate septa arranged concentrically to its surface, and recent observations by Straub confirm this view. The semi-fluid portion, as shown by H. Virchow, may be filtered away through fine muslin, leaving the tiny membranes in the residue. Straub's observations show that most of the septa arise from the region of the ora serrata, some from parts anterior to this, and others from the optic disk. There is no endothelium on the dividing membranes. In consequence of this arrangement, when an incision is carefully made in the coats of the eye so as not to divide the septa, the vitreous bulges through the wound but does not escape; if now pricked it readily escapes, a probable explanation of some of the cases of so-called fluid vitreous met with in reports.

Microscopically, the healthy vitreous shows only a scanty number of minute fibres and rounded, stellate, and spindle-shaped cells. The cellular elements are more numerous in the region of the ora serrata and of the disk.

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The posterior half of the vitreous shows scarcely any adhesion to the retina, and falls away of its own weight when a section of the eyeball is made; the anterior half is more dense and adhesive and rather strongly attached to the ciliary body.

Many of the changes to be described are probably no more disease of the vitreous than is the haziness of the aqueous in iritis disease of the aqueous humor, but are placed under this heading because the changes in the vitreous may often be the only visible sign of disease in the eye and the direct cause of the defect of sight.

Purulent Hyalitis.—Purulent infiltration of the vitreous has already been mentioned as one of the changes in panophthalmitis, but it is only in the less severe cases that this condition can be recognized clinically by the characteristic yellow reflex seen through the pupil. It is usually set up by a punctured wound in the ciliary region, with or without the lodgement of a foreign body in the vitreous, and most frequently commences within a day or two of the injury. In some rare cases it does not occur till months or years after cicatrization, reinfection of the wound having probably taken place, and Wagenmann has found cocci in the scar, the vitreous, and other tissues involved, but not *inside* the vessels, as in puerperal ophthalmitis and some other forms of purulent invasion.

The true pathological basis of purulent hyalitis is undoubtedly purulent retinitis, often combined with cyclitis, and as far as my observations go it never starts as purulent chorioiditis. The pus may in rare cases collect as a creamy fluid in the dependent parts of the vitreous, but much more often, as in Fig. 10, the process is of a more or less fibro-purulent character, the pus showing as a distinct layer only behind the lens, and elsewhere being represented by a collection of corpuscles in the branching tissue which is seen to permeate the vitreous. This condition sometimes goes on to general panophthalmitis, and even the milder cases cause total loss of sight and shrinking of the eye. Subconjunctival injection of a few drops of perchloride of mercury solution, 1 part in 500, is worth trying in an early mild case, but as a rule treatment is of no avail.

VITREOUS OPACITIES.

Opacities in the vitreous occur as fine dust, flakes and threads, larger black "floaters," or, more rarely, as membranes. They may be stationary or freely movable, according as the vitreous is of normal or of diminished consistency, and may be general or more or less localized, usually to the region of the disk or the anterior part of the vitreous. They give rise to a diminution in the acuity of the vision and to entoptic phenomena from the shadows they cast upon the retina. The vision is apt to vary at different examinations, and is usually better if a little time is allowed to permit of the opacities settling down. The reading power for print is often better than we might expect from the distant sight. One or both eyes may be affected.

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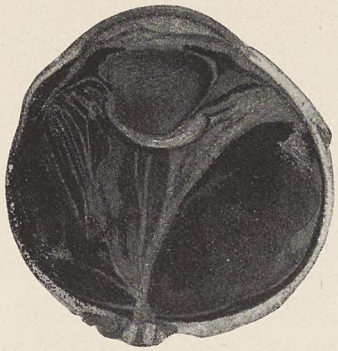
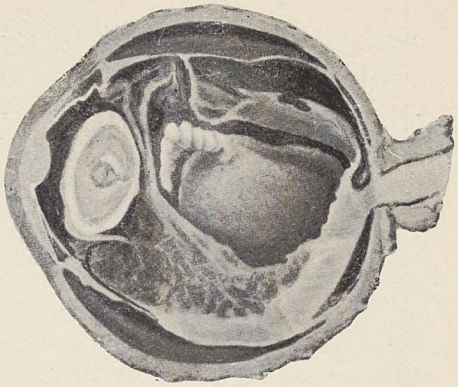
FIG. 10.



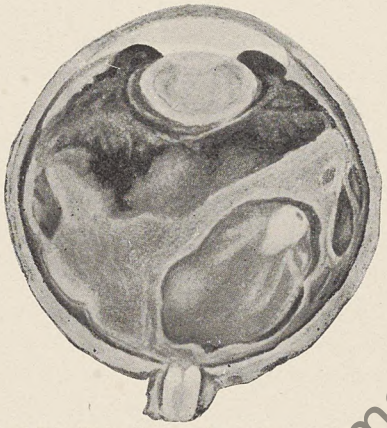
Purulent infiltration of vitreous from punctured wound of ciliary region.

FIG. 11.



Cysticercus in vitreous: neck extended, some effusion of lymph.

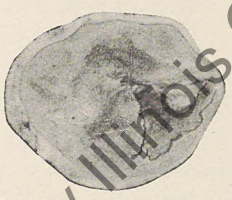
FIG 12.



Cysticercus in vitreous: head retracted, effusion of lymph around cyst, and some blood in ciliary region.

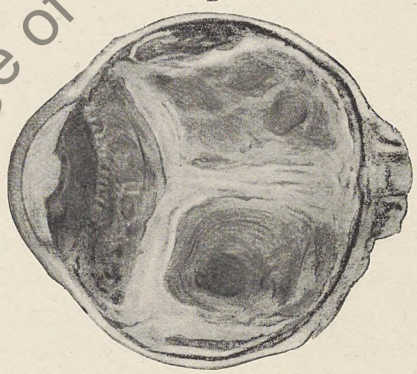
FIG. 13.

A



Sub-retinal cysticercus (twice natural size).

B



The cyst *A* has been removed from the nest-like cavity seen in *B*.

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The larger opacities are easily seen by using the ophthalmoscopic mirror alone, at about ten or twelve inches from the patient's eye, and appear as black bodies against the red background. Their detection is facilitated by dilating the pupil and causing the patient to move his eyes briskly in different directions, when they may be seen to float about.

When the opacities are very numerous and dense, little or no fundus-reflex may be obtained; but this is not often the case.

If the opacities are limited to the posterior part of the vitreous, the indirect examination will be of service, and by withdrawing the lens every part of the vitreous in the line of observation becomes successively visible as an inverted picture. In practice, however, it will be found that this method is inapplicable to the detection of opacities in the anterior parts of the vitreous, on account of the difficulty of holding the lens steady at the increased distance necessary, and under these circumstances the best way is to employ the direct examination. First of all the disk should be focussed, and then convex glasses of increasing strength are turned on, when the vitreous may be examined right up to the lens, and any opacities present become readily visible. This is by far the most delicate way of examining for the finer opacities in any part of the vitreous.

The oblique illumination can occasionally be used for the detection of opacities, but only when these are large and close behind the lens.

Speaking generally, the fine dust-like opacities in the posterior part of the vitreous are almost always caused by specific chorio-retinitis, but the same variety of opacity confined to the anterior part of the vitreous is due to serous cyclitis, which is often tubercular in origin, and may be followed by iritis. The flakes and threads are the usual accompaniment of disseminate chorioido-retinitis, and the larger black floaters are most often seen in high myopia with staphyloma and extensive choroidal disease. The membranous opacities may be caused by long-standing and severe chorioiditis, accompanied by repeated hemorrhages from the ciliary body. These may become more or less organized, and are occasionally vascular and attached to the wall of the eye at several places. Thus, they may stretch from near the disk to the ciliary region and flap about between these fixed points.

As vitreous opacities do frequently occur from visible chorioidal changes, it is only fair to assume that some, at least, of the apparently simple cases may be due to similar changes too far forward to be recognized, but in most of such cases the opacities are inflammatory or hemorrhagic exudations from the ciliary body.

Solitary black fixed opacities close behind the lens are sometimes met with on a casual examination of healthy eyes. They are probably congenital and non-inflammatory, and do not give rise to any defect of sight.

In an epidemic of small-pox, Mooren met with amblyopia and vitreous opacities without outward signs of inflammation in the eyes. Hutchinson says that gout is a frequent cause of this condition, and has met with it as the result of masturbation, and also from prolonged administration of

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arsenic. The uncomplicated cases so often seen in old people are probably correctly regarded as senile in origin. We also meet with the same condition in young people, although much less frequently, some of these going on to iritis and closed pupil and others leading to slowly forming cataract, indicating, probably, atrophic changes in the ciliary body. When the cataract has become mature, it is generally found that the eye is unfit for operation; but I have seen one case, in a boy, where removal of the lens by discission was followed by good sight.

When due to visible changes in the fundus, the treatment must be such as has already been recommended for the various forms of chorioiditis, and in all cases we must endeavor to find out and treat syphilis, congenital or acquired, tuberculosis, malaria, gout, or other constitutional disorder known to give rise to vitreous opacities.

Besides this, we may, especially in young subjects and in bad cases, have recourse to the powerful sweating effect of hypodermic injections of pilocarpine, or use saline purgatives with iron and other blood-tonics.

If seen early, often very great improvement can be obtained if the opacities are of the smaller variety, and the dust-like opacities may entirely disappear. The larger opacities, however, persist for years, and probably are permanent. We may give a more favorable prognosis when the opacities appear rapidly and occur in young subjects, active treatment here often bringing about their total disappearance,—facts which suggest their being altered extravasations of blood.

Membranous opacities appear to be quite unaffected by general treatment; they often cause detachment of the retina. When fixed at two or more points, division by means of a cutting-needle or of a very narrow knife, introduced through the sclerotic between the insertions of the external and inferior rectus tendons, may be tried, and some successful results have been recorded.

SPARKLING SYNCHYSIS.

In this affection the whole vitreous is beset with innumerable shining particles, which have been likened to fragments of gold-leaf. In many of the cases which I have seen the opacities took the form of white, glistening, round disks with the surfaces presented towards the observer, each particle shifting its position but slightly on movements of the eye, and returning to its original position when the eye became stationary. Benson records a case in vol. xiv. of the Transactions of the Ophthalmological Society of the United Kingdom, in which the vitreous was full of minute cream-colored bodies. Other observers have seen similar cases.

Sparkling synchysis is found only as a senile change, never before the age of sixty, and usually over the age of seventy. One or both eyes may be affected. Microscopical examination has determined the presence of cholesterin crystals and also of large phosphatic masses and groups of tyrosine needles. (Poncet.)

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As a rule, the sight is not affected. I have seen it normal in each eye in cases where one eye only has shown the sparkling bodies in the vitreous.

The condition is a permanent one, and no treatment is of any avail.

HEMORRHAGE INTO THE VITREOUS.

This condition is recognized by the occurrence of black or very dark masses with a red appearance at the borders. Sometimes the effusion of blood is so great that the use of the ophthalmoscope is impracticable, no fundus-reflex being obtainable, and under such circumstances the oblique illumination is of more service. In less extensive cases a view of the disk or other parts of the fundus is possible, and we may often make out hyperæmia or even actual rupture of the vessels of the disk or chorioidal changes about the equator.

The vision may fall to counting fingers or the bare perception of light, or the patient may still be able to read print, but does so in a hesitating manner, some of the words becoming obscured and necessitating his shifting the card to one side. This symptom is almost characteristic of hemorrhage into the vitreous. Patients frequently volunteer the statement that objects appear red.

The blood may become partly or entirely absorbed, but never so rapidly or so frequently as in hyphæmia. It may become organized and lead to detachment of the retina, or it may give rise to a condition, to be presently described, called "retinitis proliferans." Glaucoma and iritis not infrequently follow hemorrhage into the vitreous.

The blood may come from the vessels of the chorioid, retina, or ciliary body. I have seen the vitreous become suddenly filled with broken-up clot from the giving way of the structures in front of a large central "sub-hyaloid hemorrhage."

According to Michel, the cause may be dependent upon general disorders of the blood, as anæmia or leukæmia; disturbances of the pulmonary circulation, as in emphysema; changes in the walls of the vessels of the ciliary body from amyloid degeneration or congenital syphilis; or the hemorrhage in the vitreous may be the first sign of a tuberculosis of the ciliary body.

We meet with cases in young people where no general disorder can be made out, and the increased permeability of the vessels of the part may well be compared to the condition seen in nose-bleeders. In delayed menstruation the vitreous may become full of blood at periods of four weeks, until the establishment of the function.

Blows upon the eye, even without demonstrable rupture of the chorioid or other parts, are the causes in some cases.

Repeated hemorrhage into the vitreous, with complete or nearly complete restoration of sight in the intervals, has been noted and commented upon by Nieden, Eales, Hutchinson, and others. The patients are young men or women between the ages of twenty and thirty, and are often the

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subjects of habitual constipation or inherited gout. The tension of the eyeball in these cases is normal or diminished.

As to treatment, the internal administration of gallic acid, ergot with iron, or iodide of potassium is to be recommended, and purgatives are often of service. In addition, we may make use of subcutaneous injections of pilocarpine, keep the pupil contracted with eserine, or tap the anterior chamber at intervals.

In the case of very large traumatic hemorrhages which have resisted ordinary treatment, good results have been obtained by puncturing the coats of the eye, between the insertions of the external and inferior rectus tendons, by a broad keratome, and then giving this a half-turn so as to allow of the blood escaping.

RETINITIS PROLIFERANS.

This condition is characterized by the occurrence of glancing-white or bluish-white masses adherent to the inner surface of the retina, partially or completely concealing the disk, and from them numerous strands proceed into the vitreous, branching and interlacing, and often forming a picture of bewildering perplexity. As before mentioned, this condition is a sequence of repeated intra-ocular hemorrhage, and is due to the presence of masses of unabsorbed blood in intimate relation with the inner surface of the retina, which cause atrophy of the nervous elements and proliferation of the connective tissue, and finally become themselves transformed into fibrous tissue. Splashes of blood are often found on the retina, which is frequently detached in places.

It usually leads slowly but steadily to total blindness, and treatment is of no avail.

CHANGES IN CONSISTENCY AND VOLUME.

Apart from actual fibrous transformation, which we often see in lost eyes, one does not find increased density of the vitreous, and in many hundreds of excised eyes I have never found an increase of consistence with retention of normal transparency.

The opposite condition of diminished consistence, even to the extent of complete fluidity, is very common in high myopia with staphyloma, in chorioiditis, and as a senile change, and in the absence of opacities we may have no means of diagnosing it. When extraction of cataract is undertaken, this condition is made manifest by the escape of a quantity of watery fluid, which usually soon ceases to flow, showing that the anterior portion only of the vitreous is affected. Slight tremulousness of the iris in senile cataract is sometimes present, and gives us warning of this condition. In high myopia the fluid portion is usually confined to the posterior part.

Shrinkage of the vitreous, which may be present with fluidity or increased consistency, causes diminution in tension of the eye, and is combined with increased depth of the anterior chamber from falling back of

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the lens system. It is often accompanied by detachment of the retina, of which, indeed, according to Nordenson, it is always the immediate cause. Fluidity of the vitreous is more often combined with increased than with diminished tension.

DETACHMENT OF THE VITREOUS.

Detachment of the vitreous is almost exclusively of pathological interest. Jennings Mills found it in forty-three out of three hundred and forty-five excised globes. In the majority it occurred in the form of a triangle, with the apex at the disk and the base at the ciliary region, and more rarely as a globular mass at the back of the lens, which is to be regarded as a later stage from the giving way of the attachment to the papilla. The hyaline membrane was sometimes detached with the vitreous, at other times it remained connected with the retina, and here and there it floated in a fluid between the retina and the vitreous.

It is very doubtful if we yet know the clinical features of this condition, although Galezowski and Auguier profess to have diagnosed it in highly myopic eyes by the sudden onset of slight impairment of vision, with concentric contraction of the field of vision and a sharply defined crescentic grayish zone surrounding the papilla, within which the retinal vessels show a bend from optical conditions.

In the Transactions of the Ophthalmological Society of the United Kingdom, 1882, Swanzy records a case simulating intra-ocular growth, probably produced by hemorrhage from the ciliary body: the detached vitreous appeared as a grayish non-vascular opacity immediately behind the lens.

Persistent Hyaloid Artery.—From a clinical stand-point we class under this heading all cases which show remnants of any part of the hyaloid arterial system,—*i.e.*, the blood-vessel, its sheath, the fibro-vascular sheath of the lens, or an abnormal visibility of the hyaloid canal. Looked at in this light, the condition is by no means uncommon, but the presence of a patent blood-containing vessel is certainly one of the very rarest of ophthalmoscopic appearances. I have seen only one case, and the blood-column in this instance, being very narrow and partially concealed in the dense fibrous strand, was overlooked for some time. Perhaps a more careful and systematic examination of the cases of apparently obliterated artery might reveal a patent vessel in some of them.

A group of cases which has sometimes been erroneously classed under the term of persistent hyaloid vessel must here be mentioned. They represent in reality an abnormal course of a branch of the central artery of the retina. In these cases a vessel containing blood emerges from the disk, passes for a greater or less distance directly forward into the vitreous, and then turns upon itself to pass back to the disk close to where it emerges, the two portions forming a spiral like a miniature umbilical cord. No tissue accompanies the vessel, as in the case of the hyaloid artery. The condition usually occurs in both eyes, but may be in a very rudimentary

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state on one side, like a mere knuckle in a vessel. Pressure on the eye causes marked pulsation, the vascular strand itself also visibly jumping at each cardiac impulse. I have seen five or six cases.

In most of the recorded cases of a blood-bearing hyaloid artery this broke up in front into a system of fine vessels lying on the posterior surface of the lens and radiating towards the periphery, to carry away the blood to the ciliary regions, and, even where this is not mentioned, such a termination was probably present, as we cannot conceive how the blood could circulate in any other way. There is usually a knob-like thickening of the vessel at the posterior capsule of the lens.

A much commoner condition is the presence of a strand representing the obliterated vessel, stretching from the disk to the back of the lens. This may resemble a stout black thread with a good deal of "slack" in it, allowing of snake-like movements when the eye is rolled about, or it may be much stouter, of a glancing-bluish, tendinous appearance, thicker at each end than at the centre, and immovable.

Another variety met with is to be explained as an abnormal visibility of the hyaloid canal, and appears as a single contoured strand of delicate transparency, which on indirect examination of the disk gives an appearance as if a glass rod were interposed, but by direct examination, especially if seen end on, looks much darker.

Where no blood-column is present it is often difficult to say whether we have to do with an obliterated artery or with a visible hyaloid canal, and in some cases nothing short of a careful anatomical examination could settle the point. The latter condition is much more frequently bilateral than is an obliterated hyaloid artery, but why this should be is not known.

When an obliterated hyaloid artery occurs, as it not infrequently does, with extensive chorioidal atrophy and pigmentation in the posterior segment of the eye, we must assume that the persistence of such remains is due to the inflammatory causes at work. The strand in some cases is attached only to the disk, and much less frequently only to the lens, in the latter case being usually associated with posterior capsular opacity. Some hold, and probably with justice, that all cases of congenital posterior capsular opacity are due to remains of the hyaloid system.

Under this heading also are to be included those little patches or clumps of opaque white tissue about the centre of the disk concealing the origin of the vessels.

The remains may assume a cystic appearance, either as small, round, steel-gray bodies attached to the disk or as much longer globular formations. I have seen two remarkable examples of the latter variety, in each of which a large, bluish, cyst-like body, in shape very much like a Florence oil-flask, lay with its base adherent to the posterior pole of the eye, concealing the disk and surrounding parts for a considerable extent, the neck stretching far forward towards the lower ciliary organ. In one of the patients, a boy of seven years, there was no perception of light, and the

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defect was attributed to a severe blow on the eye; in the other, a girl of fourteen, it was said to be due to a recent illness, but the true explanation was arrived at only after watching the case for a long period and finding that no change took place.

Persistent hyaloid remains are often associated with other congenital ocular defects, as microphthalmos, hydrophthalmos, coloboma of iris and chorioid, persistent pupillary membrane, etc.

In only a small number of cases are there complaints of abnormal visual sensations, such as of shadows floating before the eye. The vision may be almost if not quite normal, or the eye may be more or less amblyopic. Most of the cases are met with on a casual examination of the eye for the estimation of refraction.

FOREIGN BODIES IN THE VITREOUS.

Foreign bodies, such as a lead pellet, a morsel of iron, copper, stone, or glass, not infrequently penetrate the coats of the eye and lodge in the vitreous, reaching this locality by going (1) through the cornea and lens, (2) through the cornea, iris, and lens, (3) through the cornea, iris, and zonule, or (4) through the sclerotic and underlying coats of the eye. In some cases the lid, usually the upper, is also perforated.

The diagnosis of such an occurrence is often a matter of considerable difficulty, and frequently we can only arrive at a probability that a foreign body has entered the eye.

The first thing we must do is to search for an opening or scar in the anterior part of the eye. If this lies in the cornea there is seldom any difficulty in detecting it, but if very small and in the sclerotic it is more likely to be overlooked from the presence of blood in, or thickening of, the conjunctiva. A good light and the use of a magnifying lens may here be necessary.

The patient's testimony, further than that he has received an injury, is seldom to be trusted. Associating, as he does, the idea of "something in the eye" with the occurrence of constant pain, he usually assures us that there is nothing in the eye, and even goes the length of declaring that he saw the piece of metal or other substance that struck the eye when we have undoubted evidence that this is actually in the eye.

Having made out the point of entrance,—let us assume that this is in the cornea and about its centre,—we find a mark on the anterior and on the posterior capsule, with a connecting streak running through the lens. If more eccentric, a hole in the iris will indicate the course of the foreign body, which in this case may reach the vitreous by going through the suspensory ligament in the circumlental space. What may by oblique illumination appear like a pigment spot or mark on the iris may often be easily made out by the ophthalmoscopic mirror to be in reality an aperture in this membrane, through which the fundus-reflex is obtainable.

By the presence of the above signs we may be quite sure that a foreign

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body has penetrated into the deep parts of the eye; but we must bear in mind that this may have gone through the globe, to be lodged in the orbit. I have seen this on several occasions with small, sharp pieces of steel and lead pellets, but have never diagnosed the condition before enucleation of the eye. I had a case in which a sharp-pointed, sword-like piece of steel over an inch in length penetrated the cornea and went clean through the eye, one end becoming firmly fixed in the bony wall of the orbit, the eye being impaled on the other end.

The foreign body may reach the vitreous directly by going through the sclerotic, in which case careful probing may be required to ascertain if the whole thickness of the sclerotic has been perforated. An opaque streak in the vitreous, one end corresponding to the wound in the sclerotic, is often visible, and indicates the track taken. When the foreign body takes this route, we are much more likely to have hemorrhage in the vitreous than when it has gone through the lens, rendering its detection more difficult; but in the latter case opacity of the lens takes place, and, if advanced, may prevent our picking out the foreign body.

The foreign body may get entangled in the coats of the eye at the point of entrance; it may lie free in the vitreous or be suspended there in a blood-clot or string of lymph; it may traverse the vitreous and become embedded in the retina; it may rebound from the back of the eye and come to rest on the posterior part of the ciliary body below; or, finally, as already mentioned, it may go through the globe into the orbit.

If the media are fairly clear, we may with the ophthalmoscope see the foreign body as a dark object with glistening margins in the vitreous or embedded in the fundus. Bubbles of air are sometimes met with in the vitreous; they are round, often multiple, glisten at the centre and appear dull and obscure at the margin, by which characters they can easily be distinguished from solid substances. They are always absorbed in from one to two days.

When searching the fundus in these cases, chorioidal plaques, often very bright and silvery-looking, give us the impression of a flake of metal lying on the fundus, but can be distinguished by their shining all over and not merely at the margins.

When the foreign body has rebounded from the back of the eye, we find a spot where the sclerotic is exposed, with some hemorrhage around it, and when present this affords us valuable information as to the nature of the accident.

Another piece of corroborative evidence is the presence of a finely speckled appearance of the macula, which is common even when the foreign body lies at a considerable distance from this part of the eye.

Chemically indifferent and smooth objects, such as a piece of glass, a lead pellet, or a portion of highly polished wire, may remain a long time in the vitreous without causing any inflammation; but a fragment of iron with roughened surface soon gives rise to inflammation and becomes com-

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pletely encapsuled in lymph in about a fortnight, after which the inflammation subsides, but is very apt to recur.

Rusty discoloration of the iris, lens, and other tissues takes place from retention of a piece of iron, and in long-standing cases where the media are opaque this sign is of considerable diagnostic appearance.

In all operations for the removal of a foreign body from the eye strict antiseptic precautions should be used, and the patient must be deeply anæsthetized.

When the body is not magnetizable, our chance of successfully removing it is so very small that this should be attempted only if we can actually see it, and it happens to lie near the equator, even in the superficial layers of the vitreous or in contact with the coats of the eye. Under these circumstances an incision is made by a lance-thrust, when gentle pressure may cause the foreign body to escape along with a slight prolapse of vitreous, or, keeping it in view by the ophthalmoscope, a forceps may be introduced and the foreign body seized and removed. Unfortunately, however, it frequently happens that our attempts are fruitless: the vitreous escapes, but the foreign body remains in the eye.

Before undertaking the operation, it is advisable to get the patient's permission to remove the eye at once if we should not succeed in extracting the foreign body.

If it is a piece of iron or steel that has entered the eye, our chances of success are much greater, even if it cannot be seen, for we can use the electro-magnet; but even here we are likely to succeed only if the operation be undertaken within a day or two of the accident, before it becomes covered with lymph, and if it is not embedded in the coats of the eye.

If the wound of entrance is in the sclerotic, this should be enlarged, and the electric needle thrust into the vitreous towards the piece of metal, if seen, or in the direction in which it is supposed to lie. If successful, a "click" is both heard and felt, and on withdrawing the instrument the foreign body will be found attached. The conjunctiva is then sutured over the wound in the sclerotic. Very little if any reaction follows this procedure, which may be done with advantage, even if we cannot be sure that there is anything in the eye.

If the foreign body has gained the vitreous by going through the cornea, and the lens is clear or only slightly opaque, our best plan is to dissect up a conjunctival flap about the equator towards the outer side of the inferior rectus, and make an incision through the coats of the eye at least five or six millimetres in length in a sagittal direction. The most suitable instrument for this purpose is a sharp Graefe's knife, and it should be thrust deeply into the vitreous to reach the foreign body if possible, which renders the use of the magnet much more effective. It is not a good plan to wait for the lens to become opaque in order that we may get at the foreign body by the incision used for the extraction of the lens, for any advantage we thus gain is greatly overbalanced by the piece of metal becoming coated with

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lymph and more or less fixed. It is astonishing how slight an amount of fixation will render the use of the magnet ineffectual. I have frequently demonstrated this on opening an eyeball containing a piece of metal, which may readily jump at and become attached to the magnet, but as soon as the string of lymph is put on the stretch by withdrawal of the magnet the fragment springs back to its original position. When an operation is considered advisable, it should be done at once.

If the lens is already opaque when we first see the case, the relative positions of the scars in the cornea and lens-capsule give us a probable idea as to the direction the piece of metal has taken. The presence of a blank in the field of vision, taken by means of a lighted candle or by throwing a beam of light on the eye from different directions, may also be of use. Tenderness localized to one particular part of the ciliary region is spoken of, but personally I have never found it of any service in locating a foreign body.

In most cases, however, we can form no idea where the foreign body is, and must be content to assume that it lies, as is usually the case, somewhere in the lower part of the vitreous. The lens ought to be removed by an ample lower corneal flap with iridectomy, and the electro-magnet introduced well into the lips of the wound. If by these means we fail to remove the foreign body, we should desist from further efforts for the time, bandage up both eyes, and let the patient be kept quiet in bed. The wound usually heals almost as quickly and satisfactorily as after an uncomplicated cataract extraction, and when the eye has quite recovered we may be able to see where the foreign body lies, although by this time it is almost sure to have become covered up with lymph. If it can be seen, whether covered with lymph or not, another attempt at removal by the electro-magnet should be made, through either a corneal or a scleral incision, as may seem advisable, and, assisted as we are by knowing its exact position, our efforts may be crowned with success.

If again unsuccessful, and the eye keeps irritable, we should certainly enucleate; and even if there be no irritation it is well to do so if the patient cannot be kept under observation.

If after the removal of the lens and the first unsuccessful magnet operation the state of the eye after recovery permits of a good view of the fundus and yet no foreign body can be seen, we may be pretty sure that this lies on the posterior part of the ciliary body below,—a site which, as mentioned before, is a very common one. From what I have myself seen, I can state that the electro-magnet is here of no use whatever. I have frequently found a foreign body in this position in excised eyes, where the magnet must have been repeatedly brought in contact with it. I believe that the method which Leber employs in the case of fragments of copper or other non-magnetizable substances fixed in this position is the only way in which we are likely to succeed. He makes two incisions completely through the coats of the eye, which diverge as they pass backward into

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the ciliary region, and then everts the triangular flap thus formed, on the inner surface of which the fragment of metal is often found and may be removed with forceps.

After the extraction of the foreign body, Leber makes culture preparations with the exudation around it. If these prove the absence of micro-organisms, he continues with conservative treatment; but if organisms are present, he enucleates the eye, on account of the great danger of sympathetic inflammation. This rule he has followed for many years, and has never had reason to regret doing so.

When the foreign body can be seen embedded in some part of the fundus and is causing no irritation, it is not advisable to attempt its removal. Such eyes often keep quiet and retain good sight. In spite of what is often asserted, there is very little if any fear of sympathetic inflammation, for the reason that such small foreign bodies penetrate the eyes aseptically.

When the eye has been penetrated by a very large piece of metal, weighing two hundred milligrammes or more, the case may be regarded as hopeless from the first, and the eye should be at once removed.

PARASITES IN THE VITREOUS.

Only two kinds of parasites have been described in this situation,—the cysticercus cellulosæ, fairly common in certain countries, and a species of filaria of extreme rarity. We must add one more to this group, for I have seen an undoubted case of hydatid cyst in the vitreous, which, with some cases of cysticercus, is recorded in vol. xvii. of the Transactions of the Ophthalmological Society of the United Kingdom, to the Council of which Association I am indebted for permission to make use of reproductions of the figures in the paper referred to.

Most of the cases of "filaria" have in all probability been persistent hyaloid arteries: the first certain example of filaria was recorded by Kuhnt some ten years ago, in a man aged thirty-one, in whose right eye was seen a sharply contoured glancing-white bladder about the size of the optic disk. It increased in size, got darker in color, and was removed through a scleral section. The parasite, 0.38 millimetre in size, was contained in a little gray lump, and was at once recognized as a filaria by Leuckart, to whom it was given for an opinion.

Cysticercus in the vitreous appears to be an exceedingly rare occurrence in Great Britain, and probably very few ophthalmic surgeons in this country have seen a single case. De Wecker, in France, has seen one case among sixty thousand eye-patients; Mauthner, in Austria, has not found one in thirty thousand; while in North Germany, where raw meat is a common article of diet, it occurs as often as one in one thousand.

We have seen six in one hundred and eighty thousand patients, or one in thirty thousand. None of our cases were found in foreigners.

It appears that both eyes are never affected, but two parasites have been met with in one eye.

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In one case hundreds of little pearl-like growths containing cysticerci were visible under the patient's skin, but it is exceptional to have coincident evidence of the disease apart from the eye. In one of our cases the use of an anthelmintic brought away a tape-worm.

The parasite is most frequently met with between the chorioid and the retina. It may remain in this situation or may perforate the retina and reach the vitreous. It may gain the vitreous directly by way of the vessels of the retina or of the ciliary body.

In the two cases of living cysticerci in the vitreous which have come under my observation, the ophthalmoscope showed a large, spherical, bluish-white cyst, and, springing from this, the neck of the animal, like an alabaster pillar, surmounted by the head and suckers, which, with its slow, regular, and graceful movements, reminded one of an elephant's trunk, the whole appearance presenting a picture which, once seen, could never be forgotten.

In the early stage, when the animal is living, there is no difficulty whatever in diagnosing the affection; but when it has been dead for some time and effusion of lymph has taken place, great difficulty may be experienced in arriving at a correct opinion.

When situated beneath the retina, the cyst appears as a bluish-white, sharply defined body with yellowish, shimmering border, over which the retinal vessels course uninterruptedly. The retracted head is indicated by a light spot on the cyst. Peristaltic movements of the entire cyst may be seen, and when present are characteristic.

Wherever situated, vitreous opacities come on gradually and increase in density, and the cysticercus becomes enveloped in a connective-tissue capsule. Circumscribed inflammatory changes take place in the chorioid and retinal pigment layer, and shrinking of the eye takes place, seldom accompanied by formation of pus. The changes in the interior of the eye resemble those caused by the lodgement of a foreign body.

As shrinking and loss of the eye invariably take place unless we succeed in removing the parasite, this should always be attempted, and is done in the following manner.

The eye is well cocainized, or the patient is chloroformed; the strictest antiseptic precautions must always be employed. By means of a suture passed through the subconjunctival tissue close to the cornea, the eye is strongly rotated in a direction opposite to the situation of the cysticercus. A conjunctival flap is dissected up from the equatorial region, and, after the bleeding has ceased, a meridional incision about one and a half centimetres in length is made through the sclerotic by the thrust of a von Graefe's knife held with its back towards the eye. The cyst in many cases presents in the wound and is easily removed, or we may have to separate the lips of the incision and extract the cyst by forceps or a small hook. When possible, we should lay hold of the neck rather than of the body, as the former is least likely to give way. The conjunctival wound is then sutured, and the eye closed with a firm antiseptic bandage. This operation

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has in recent years been twice done at the hospital with which I am connected, and in each case successfully. Those who have a large experience reckon their successes at from sixty to seventy per cent.

When the cysticercus is stationary, and lies between the retina and the chorioid, it is much more easily dealt with than when it is freely movable and situated in the vitreous. If the eye is shrinking, and especially if, in addition, it is inflamed and painful, it is far better to enucleate the globe than to attempt to remove the parasite.

If the case comes under observation after the formation of a connective-tissue capsule around the parasite, we shall find a large, bluish-white, rounded mass of lymph like a ball of cotton wool, and I have come to the conclusion that such a condition, in the absence of a puncture-wound or a foreign body in the eye, is sufficient evidence of the nature of the case.

Hydatid cysts are occasionally found in the orbit, causing proptosis; but, so far as I know, the case I am about to describe is the only example of such a cyst occurring in the eye itself.

The patient was a healthy little girl three years and eight months old, who was brought to the out-patient department under my care for what looked, on a casual examination, like some form of posterior capsular cataract in the right eye.

The sight was gone, the eye was free from injection, and the tension was normal. A closer examination showed a dense white, glistening, non-vascular opacity in contact with the back of the clear lens. It did not present a concave surface like a posterior capsular cataract, but was quite flat.

No free edge could be made out in any direction, even on wide dilatation of the pupil with atropine, nor was there a trace of fundus-reflex to be seen at any part. Although the iris was not advanced, the opacity looked distinctly nearer to the cornea than an opacity behind the lens ought to look.

When I saw the child in the hospital a few days later, after the use of atropine, glaucoma had taken place, the eye was injected and very painful, and the cornea was slightly hazy. We removed the eye, as it was thought that there might be an intra-ocular growth.

The interior of the organ was lined by a dense, continuous membrane which was closely adherent to the lens, ciliary body, and retina and occupied accurately the position of the hyaloid membrane.

The lens was diminished in thickness at the expense of the posterior half, the posterior surface being almost flat,—a state of affairs which satisfactorily accounted for the undue proximity of the opacity to the cornea noted before excision. Microscopic sections of the entire globe in the horizontal plane showed that the chorioid, retina, and other parts were practically normal. The cyst-wall was composed of numerous superimposed, structureless lamellæ. Some of the most internal of these had in places become detached at one end, and curved inward like a watch-spring or the shavings of wood from a carpenter's plane.

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THE ANATOMY OF STAPHYLOMA POSTICUM, AND THE RELATIONSHIP OF THE CONDITION TO MYOPIA.

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I. Ever since the early part of the present century, when Scarpa described staphyloma of the posterior segment of the sclera, this anomaly, which he accidentally discovered in two eyes taken from cadavers, has been known as the posterior staphyloma of Scarpa. Scarpa's description was confined to the anatomical relations of the condition,[1] as he was able to learn nothing concerning the clinical symptoms attending its initial stages and subsequent progress.

Many years after Scarpa's discovery, Ammon published an account of the post-mortem appearances of two eyes with posterior staphyloma,[2] but he could give no information concerning the functional disturbances attending this anomaly of form. He expressed the opinion, however, that it was by no means so rare as was then supposed, and called attention to the striking resemblance it bore to the scleral protuberance of the fœtal eye, which he had previously described. He also emphasized the fact that of all parts of the sclerotic, that situated at the central part of the fundus was most liable to thinning and stretching, for the reason that in the human eye firm union does not take place at this point until after birth.

Ammon was the first to attempt to connect the development of posterior staphyloma with the antecedent stages of its evolution. It is surprising that it did not occur to him that it might occasion near-sightedness, and also that Ritterich, who was aware that the eyes of a man in whom he found the condition had always been very myopic, does not appear to have recognized the relationship between myopia and posterior staphyloma,[3] especially as

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[1] Trattato delle principale malattie degli occhi di A. Scarpa, 1816, vol. ii. p. 146.

[2] Zeitschrift für Ophthalmologie, Bd. ii. S. 247; Ammon. de genesi et usu maculæ luteæ. Vinariæ, 1830.

[3] Schmidt's Jahrbücher, 1842, Bd. xxxvi. S. 138.

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Beer had already pointed out that myopic eyes were often relatively large and long, and that abnormal length of the eyeball was the most frequent cause of myopia.[1]

It was, however, reserved for Arlt to recognize the connection between the posterior staphyloma of Scarpa and myopic refraction, and it was this author who first announced that the condition was the regular anatomical foundation of typical myopia.[2]

At the time that Arlt made this important announcement, the ophthalmoscope was coming into use in the examination of myopic eyes, and conus was seen to be uniformly present in them. This discovery led to the general acceptance of Arlt's opinion. It was regarded as established that conus was the evidence of an atrophy of the chorioid, resulting from distention of the posterior segment of the eyeball,—that, in other words, conus and staphyloma were invariably associated; the assent generally accorded to this view being indicated by the facts that conus was also known by the name of staphyloma, and that posterior staphyloma of Scarpa was at once diagnosed if a conus were seen to be present. This belief in the necessary interdependence of conus and posterior staphyloma was so firmly established that, on account of the occurrence of coni in emmetropic and hypermetropic eyes, the presence of posterior staphyloma of the sclera in eyes that were not myopic was regarded as a matter of course, although the existence of posterior staphyloma in emmetropic and hypermetropic eyes could never be proved by anatomical investigation. Myopia and posterior staphyloma became, as Donders expressed it,[3] "almost synonymous terms," and remained so for several decades.

It was not until the year 1883 that Tscherning[4] proved, in an able criticism, that this usage had no scientific justification, and four years later Stilling[5] expressed the opinion that the majority of myopic eyes had normal scleræ and normal or but slightly elongated axes, and were therefore entirely free from posterior staphyloma even though coni might be present.

II. The preceding sketch of the growth of our knowledge concerning the connection between posterior staphyloma and myopia shows that the general acceptance of posterior staphyloma as the anatomical basis of myopia was founded upon (*a*) facts gleaned from descriptions of myopic eyes after death, and (*b*) the interpretation of the ophthalmoscopic appearances presented by myopic eyes during life.

We must now examine each of these sources of information.

(*a*) Arlt based his opinion upon the anatomical examination of eight myopic eyes. Stilling, supported by the results of the examination of ten myopic eyes, opposed Arlt's views. Of the eight eyes examined by Arlt,

[1] Beer, Lehre von den Augenkrankheiten, Wien, 1817, Bd. ii. S. 654.
[2] Arlt, Die Krankheiten des Auges, Bd. iii. S. 240.
[3] Die Anomalien der Refraction und Accommodation des Auges, S. 296.
[4] Studien über die Aetiologie der Myopie, Archiv für Ophthalmologie, xxix. 1, 252.
[5] Untersuchungen über die Entstehung der Kurzsichtigkeit, S. 119.

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six presented a high degree of myopia and two a myopia of about three diopters. Of the ten eyes used in Stilling's investigations, seven had myopia of high degree and three of moderate degree. The thirteen highly myopic eyes examined by these two authors had posterior staphyloma. The five eyes with only a medium degree of myopia, without exception, showed no evidence of posterior scleral staphyloma. The conclusions arrived at by the two authors were contradictory, although the appearances they described were similar.

Their observations showed that eyes with a medium degree of myopia were of the same length as, or but a trifle longer than, emmetropic eyes, and that their scleræ were normal in color and normally resistant, while eyes with a high degree of myopia were characterized by the presence of posterior staphyloma.

Of thirty-four myopic eyes anatomically examined by Herrnheiser and myself,[1] twenty-one had a myopia of at least ten diopters, and thirteen a myopia of from two to eight diopters. Of the twenty-one highly myopic eyes, twenty had posterior staphyloma; of the thirteen with low or moderate myopia, not one had posterior staphyloma. The eyes with high myopia were not less than twenty-seven and not more than thirty-two millimetres in length; of the eyes with from two to eight diopters of myopia, nine were not less than twenty-three and not more than twenty-five millimetres in length, one was twenty-five and five-tenths millimetres long, two twenty-six millimetres, and one twenty-seven millimetres.

The total number of myopic eyes examined by Arlt, Stilling, and the writer aggregates fifty-two. Thirty-four of these were highly myopic, and eighteen had a myopia not exceeding eight diopters. In the group of highly myopic eyes there was but one without posterior staphyloma, while in the second group not a single staphyloma was found.

If we supplement this summary of the descriptions of the form of myopic eyes hitherto published by the mention of two eyes each reported by Weiss[2] and Krotoschin,[3] with M. 5 and M. 6, but no posterior staphyloma, and by the facts that five highly myopic eyes examined by Donders, varying in length from twenty-eight and five-tenths to thirty-three millimetres, were all affected with posterior scleral staphyloma, and that many other authors who have had opportunity to examine anatomically eyes with high myopia always found deformity from posterior staphyloma present, it may be confidently asserted that by far the greater number of myopic eyes, especially those having myopia of low or medium degree (the eyes which usually acquire their myopia at school), have absolutely no posterior staphyloma, but that this condition is found only in eyes (fortunately rarely met with) that have a myopia exceeding ten diopters. The correctness of this propo-

[1] Ueber Staphyloma posticum, Conus und Myopie, Zeitsch. f. Heilkunde, Bd. xvi.

[2] Beiträge zur Anatomie des myopischen Auges. Nagel's Mittheilungen aus der ophthalmologischen Klinik in Tübingen, iii. Heft.

[3] Archiv für Augenheilkunde, Bd. xxi. S. 33.

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sition can scarcely be questioned, notwithstanding the remarkable assertions of von Jaeger. This author, who has contributed so much to the knowledge of myopia, reports the existence of posterior staphyloma with M. 1/24 and axial length of twenty-five and seven-tenths millimetres, with M. 1/12 and axial length of twenty-seven and seven-tenths millimetres, with M. 1/10 and axial length of twenty-eight millimetres, with M. 1/8 and axial length of twenty-nine and seven-tenths millimetres, with M. 1/7 and axial length of thirty millimetres, with M. 1/6 and axial length of thirty and three-tenths millimetres, and with M. 1/5 and axial length of thirty-one millimetres.[1] The writer's experience of the length of eyes with from M. 3.5 to M. 8, and of the degree of myopia associated with an axial length of from twenty-seven and seven-tenths to thirty-one millimetres, is irreconcilable with von Jaeger's statement. The writer does not doubt that eyes with an axial length of twenty-seven and seven-tenths, twenty-eight, twenty-nine and seven-tenths, thirty, thirty and three-tenths, and thirty-one millimetres respectively were affected with posterior staphyloma, but he can scarcely believe that the myopia of these eyes amounted to only 1/12, 1/10, 1/8, 1/7, 1/6, and 1/5. Notwithstanding the reasonableness of this doubt, the statement of von Jaeger must not be disregarded, and from the results of the investigations of all authors it can only be assumed that posterior staphyloma is, as a rule, present in the comparatively few eyes that have M. 10 and over, and only exceptionally in the extremely numerous instances of eyes that have from M. 2 to M 8.

(*b*) Tscherning examined the refraction of seven thousand five hundred and sixty-four men from eighteen to twenty-five years of age, from all classes of society, and found among them six hundred and twenty-seven with myopia of 2 D. and over. Of these six hundred and twenty-seven myopes, fifty-one (eight per cent.) had more than 9 D. of myopia. From what has been previously said, we may assume that this eight per cent. had staphyloma. Tscherning states that two hundred and ninety-one of his myopes, or forty-six per cent., had coni. (Small coni that could not be differentiated from a wide scleral border were not included in this summary.) Of the two hundred and ninety-one cases presenting coni, two hundred and twenty-four had from M. 2 to M. 6; thirty-one, from M. 7 to M. 9.

As Arlt, Weiss, Stilling, Krotoschin, and Herrnheiser and the writer did not find a single posterior staphyloma among nineteen eyes with from M. 2 to M. 6, and as in thirteen of these eyes the axial length varied between twenty-three and twenty-five millimetres, just as in emmetropic eyes, in six reaching at most twenty-six millimetres, we are justified in concluding that among the two hundred and twenty-four pairs of eyes with from M. 2 to M. 6 in which Tscherning found coni, none were affected with posterior staphyloma. If we also do not take into account the thirty-one subjects with from M. 7 to M. 9 in whom Tscherning found coni, and consider only

[1] Ueber die Einstellungen des dioptrischen Apparates, 1861, Ss. 262 bis 269.

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that he found coni in two hundred and twenty-four subjects with from M. 2 to M. 6, and in thirty-six individuals with more than 9 D. of myopia, it follows that among the two hundred and sixty myopes with coni only thirty-six (*i.e.*, fourteen per cent.) had posterior staphyloma.

If Tscherning had included the cases of M. that were less than 2 D., and had not disregarded the smaller coni, the percentage of cases with conus in which posterior staphyloma was present would have proved considerably smaller.

As we are here endeavoring only to show how erroneous the custom is of inferring the existence of a posterior staphyloma from the presence of a conus, we may content ourselves with the conclusions drawn from Tscherning's figures. We must not, however, omit to add that when coni were found in eyes that were not myopic, the presence of posterior staphyloma was also inferred, in spite of the fact that this condition had never been found in an emmetropic or a hypermetropic eye. Of one hundred and thirty-five eyes with coni the writer has found ninety-nine to be myopic, eighteen emmetropic, and eighteen hypermetropic.[1] As it is certain that not a single non-myopic eye with conus has posterior staphyloma, and as of myopic eyes (even with Tscherning's estimate as a basis of computation, which is not entirely correct so far as the frequency of conus in myopic eyes is concerned) only fourteen per cent. present this anomaly, it is evident that of the total number of eyes with coni only a small proportion (about 1/20) has posterior staphyloma.

Of thirteen eyes with from M. 2 to M. 8 examined anatomically by Herrnheiser and the writer, nine had conus, but not one posterior staphyloma; of twenty-one eyes with from M. 10 to M. 20, only one had no staphyloma and but two no conus. From this it follows that, although conus is a regular concomitant of an existing staphyloma, yet staphyloma is only exceptionally to be inferred from the presence of a conus. In other words, when in the anatomical examination of a myopic eye a staphyloma is found, one may almost certainly expect to find a conus; when, however, the presence of a conus has been recognized by the ophthalmoscope in the examination of a myopic eye during life, the existence of a posterior staphyloma may be expected only when the myopia amounts to at least 10 D.,—that is to say, in a small percentage of cases.

III. Since conus was first recognized it has been regarded as a circumscribed atrophy of the chorioid. Arlt wrote in 1876, "This alteration [the conus] must, from an anatomical point of view, be described as an atrophy of the chorioid, its remoter cause being stretching of that membrane."[2] In some eyes with conus, in those, for example, having posterior staphyloma, the chorioid is, as a matter of fact, thin in the entire posterior

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[1] Zur Lehre von den Ursachen der Kurzsichtigkeit, Archiv für Ophthalmologie, xx. 2, 40.

[2] Ueber die Ursachen und die Entstehung der Kurzsichtigkeit, 1876, S. 6.

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segment of the globe, and spread over a greater surface than in the normal eye. It is reasonable to assume that the chorioid in such eyes has undergone stretching. In by far the greater number of eyes with conus (in those which have normal length and shape) the chorioid beyond the edge of the conus is normal. The conus in these eyes is, nevertheless, similar to the conus that is found in eyes with posterior staphyloma. As in the former the chorioid is not stretched, the conus cannot be regarded as an atrophy from stretching, whether the eyes in which it occurs have staphyloma or not. Von Jaeger found that the chorio-capillaris was sometimes absent within the limits of the conus, and that in extensive staphylomata the chorioid over the conus presented the appearance of a glass-like, homogeneous membrane which was exceedingly fine and delicately striated and contained a few vessels.[1] This description certainly tends to corroborate the correctness of the assumption that the chorioid is atrophic within the limits of the conus, although it may not have become so in consequence of stretching.

These anatomical changes in the chorioid are, however, not the only ones present in conus. They alone fail to explain the ophthalmoscopic appearances and the functional disturbances at its site. They might all be present without constituting a conus if the pigment epithelium and the layer of rods and cones were not also wanting over the area embraced by the defect. The essential pathological changes (those that are peculiar to conus, and without which it could not exist) are situated in the retina, and not in the chorioid. The absence of the pigment epithelium in a certain part of the fundus, usually of crescentic form and always immediately adjoining the outer edge of the papilla, and the absence of light-perceiving elements within this area (which, therefore, is a part of the blind spot), are the features of conus that render it, in position, shape, and function, a clearly defined anomaly. In these particulars conus differs greatly from the atrophic chorioidal patches seen in the fundus, with which it has color in common; and its true nature is misrepresented if nothing more be said of it than that "it must be regarded as an atrophy of the chorioid."

Nevertheless, the belief that conus was merely a circumscribed atrophy of the chorioid obtained wide-spread acceptance until Stilling, in the year 1887, cast some doubt upon it by his endeavor to prove anatomically that the white crescent or ring, as the case might be (the conus), was not situated in the ectatic chorioid, and could not therefore result from atrophy of that membrane.[2] The writer has examined nineteen coni anatomically, five of them annular and fourteen crescentic, and has arrived at the following conclusions concerning the anatomical foundation of the conus:

(*a*) In eyes with annular conus the diameter of the sclero-chorioidal canal and of the part of the optic nerve surrounded by it is unusually

[1] Ueber die Einstellungen des dioptrischen Apparates, S. 60.

[2] Untersuchungen über die Entstehung der Kurzsichtigkeit, S. 125.

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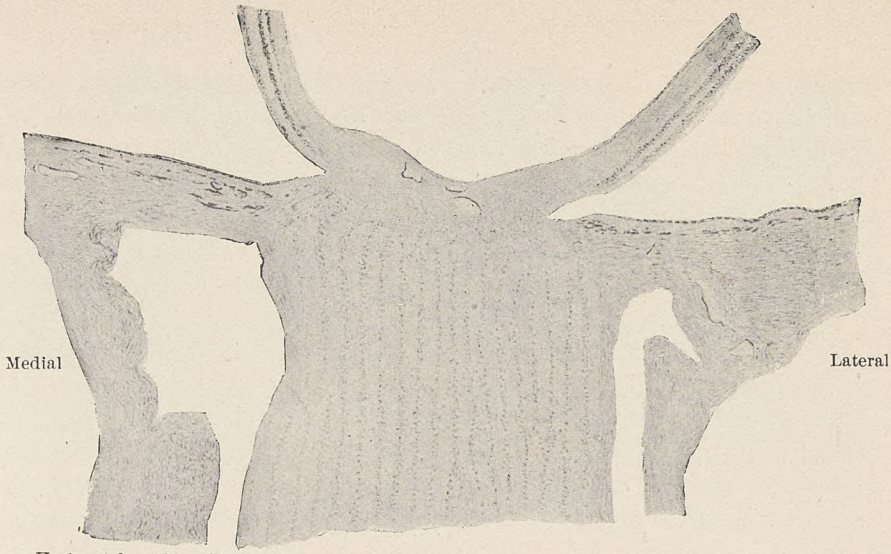
FIG. 1.

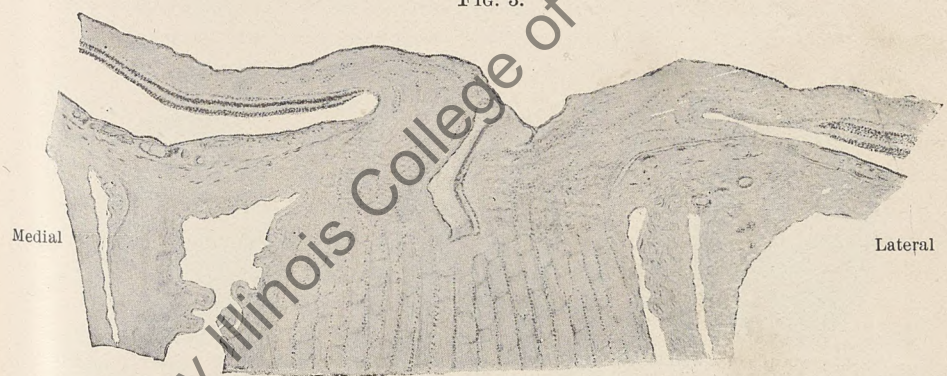


Horizontal section of the optic nerve of a man fifty-two years of age, showing a narrow annular conus. M. 2 D. Length of axis twenty-five and a half millimetres, horizontal diameter twenty-six millimetres, vertical twenty-five millimetres. No posterior staphyloma.

FIG. 2.

Vertical section through the optic nerve of the eye of a man forty-three years old, with a large annular conus. M. about 20 D. Length of axis thirty millimetres, transverse diameter twenty-seven and a half millimetres, vertical twenty-five millimetres. Large posterior staphyloma.

FIG. 3.



Horizontal section through the optic nerve of an eye with a large posterior staphyloma and a crescentic conus. Length of axis twenty-nine millimetres, horizontal diameter twenty-six and a half millimetres, vertical twenty-five millimetres. Degree of myopia unknown.

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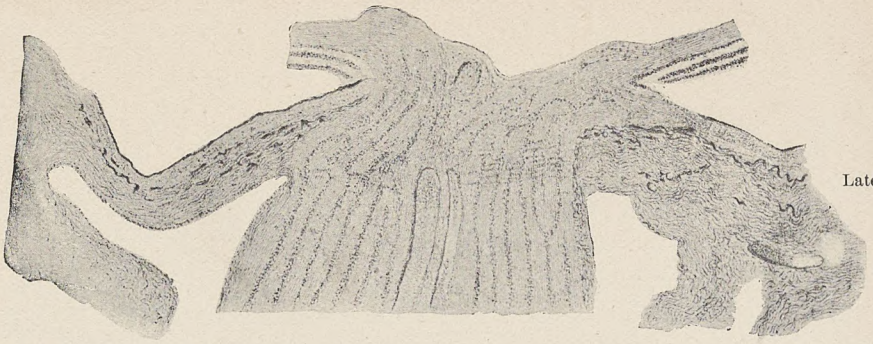
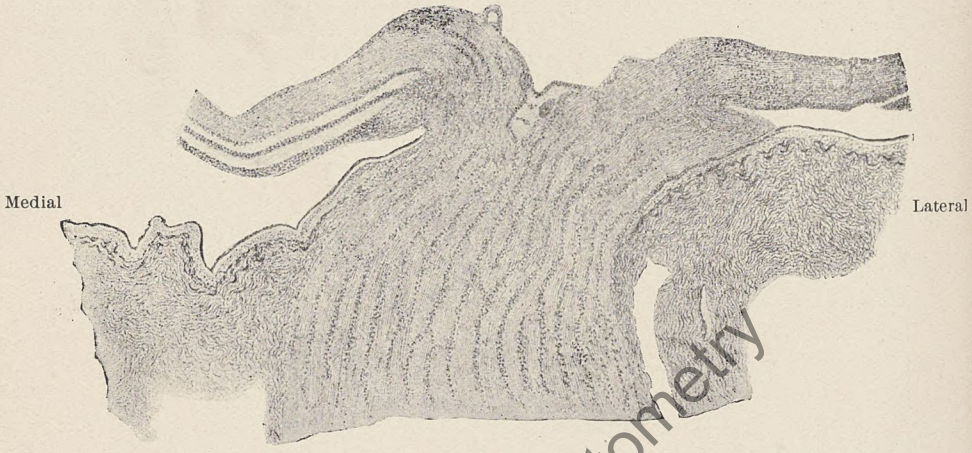
FIG. 4.



Horizontal section through the optic nerve of the eye of a woman twenty-eight years old, with a crescentic conus three-fourths of a papilla-diameter in width. M. about 20 D. Length of axis twenty-nine millimetres, transverse diameter twenty-seven, vertical twenty-four millimetres. Posterior staphyloma. The intervaginal space on the inner side is very wide and of an unusual form.

FIG. 5.



Horizontal section through the optic nerve of the eye of a woman thirty-seven years old, having a crescentic conus of greater width than the papilla. M. 13 D. Length of axis twenty-seven millimetres, horizontal diameter twenty-five, vertical twenty-five millimetres. Posterior staphyloma. The papilla was vertically oval, an appearance explained by the so-called "supertraction" of the chorioid on the nasal side.

FIG. 6.

Myopic eyeballs of a woman twenty-three years of age. Between them, for the sake of comparison, there is placed an emmetropic eyeball of a woman thirty years old. The myopic eyes were shaped like cylinders with rounded ends. The right eye was twenty-eight and one-half millimetres long, the left was thirty millimetres. The degree of the myopia of the right eye could not be measured, that of the left amounted to 20 D. The emmetropic eye was twenty-four millimetres long.

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large; the diameter of the papilla, however, is normal. When the intra-chorioidal portion of the optic nerve has a diameter of, for example, three millimetres, and the intra-retinal portion (*i.e.*, the papilla) a diameter of one and five-tenths millimetres, a white zone is seen between the edge of the papilla and the peripheral portion of the connective tissue (or of the chorioidal ring, as the case may be); and it is this that forms the annular conus observed by the ophthalmoscope. The annular conus, then, is not situated in the region of the ocular membranes, but in that of the optic nerve,—its white color coming not from the sclera that has become visible, but from the lamina cribrosa. The peripheral limits of the conus are formed by the chorioidal ring. As the layer of pigment epithelium begins at this ring, there can be no pigment epithelium within the area included by the annular conus. The pigment epithelium has not disappeared; on the contrary, it has never existed in the part of the fundus in which the conus is situated.

The annular conus forms a part of the transverse section of the optic nerve seen by the ophthalmoscope, and belongs, therefore, in its entire extent to the blind spot. The chorioid over the staphylomatous area is thinner than normal, but the part of it immediately surrounding the optic nerve does not differ from the part situated behind the fovea centralis or the parts which cover the other portions of the scleral staphyloma. It is, therefore, out of the question that the stretching of the chorioid due to stretching of the sclera could occasion chorioidal atrophy of such a degree as to be scarcely distinguishable from the absence of that membrane, in the vicinity of its insertion. As the tunics of the eye affected with posterior staphyloma of Scarpa have a much greater superficial extent than those of the normal eye, the chorioid over the area of the staphyloma is thinner than in the normal eye. There is, however, no marked circumscribed atrophy of this membrane, no entire disappearance of the pigment epithelium, and no loss of the percipient layers of the retina limited to a sharply defined region around the papilla. The changes in the chorioid and retina which have been regarded as the anatomical basis of the annular conus, and which have been so zealously explained, do not exist; the annular conus being due to an anomaly in the form of the optic nerve resulting from structural irregularities in the external tunics of the eye that are characterized by deficient development of the sclera and chorioid around the optic nerve, shown in the abnormal width of the sclero-chorioidal canal at the entrance of the optic nerve.

(*b*) The crescentic conus in the myopic eye with posterior staphyloma has the same anatomical foundation as the crescentic conus in the myopic eye of average length and form. The conus occurring in myopic eyes in which the chorioid of the posterior half of the globe is abnormally thin presents the same structure as in myopic eyes in which the chorioid from the edge of the conus to the ora serrata is normal. Within the limits of the crescentic conus the chorioid is invariably abnormal. The anterior

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layers of this membrane, the elastic lamina and the chorio-capillaris, are here absent, while the posterior layers are thinner than normal. They are also faintly or not at all pigmented, and contain few or no vessels. Over the anterior surface of the conus, and intimately united with it as far as its edge, is spread the most anterior part of the periphery of the pial sheath of the optic nerve. In front of this lie fibres of the optic nerve, which have bent over in the plane of the ocular membranes with the pial sheath, which they accompany to the edge of the conus, where they turn back towards the axis of the optic nerve, to reach the nerve-fibre layer by way of the papilla. Since within the limits of the conus the pial sheath and the nerve-fibres lie immediately upon the anterior surface of the chorioid, the retina cannot begin until the periphery of the conus is reached, and therefore there can be no pigment epithelium upon the surface of the conus, which consequently must belong to the blind spot. The pigment epithelium could not have disappeared from within the conus, as it has never been present in this situation. Neither have any changes taken place in the percipient layers of the retina within the area of the conus which could have resulted in an enlargement of the blind spot proportionate to the area of the conus; for the retina, never having been present in front of the conus, could necessarily never have undergone change at that point.

Absence of the elastic lamina within the area of the conus cannot be regarded as an atrophy of that membrane, because the pial sheath of the optic nerve spreads over the defect in the elastica and unites with it at its periphery, just as in the normal eye. Neither can the complete disappearance of the elastic lamina and of the chorio-capillaris be given as the reason why the pial sheath and the nerve-fibres lying upon it leave their normal position and extend into the defect in the anterior layers of the chorioid. As the anomalous position of the pial sheath and the fasciculus of optic nerve fibres form a constant anatomical feature of the crescentic conus, the defect in the anterior layers of the chorioid can only be regarded as a structural anomaly.

It must be remembered that the pial sheath in every eye accompanies the optic nerve to the point of its passage through the chorioid, and unites in the anterior plane of that membrane with the border of the elastic lamina of the chorioid. If the elastic lamina does not extend to the edge of the chorioidal canal, the pial sheath, in order to join it, must bend over at the edge of the chorioidal canal in the plane of the ocular membranes and extend across the anterior surface of the chorioid as far as the edge of the elastic lamina.

As the retina always begins at the outer edge of the crescentic conus, and as the fasciculus of optic nerve fibres and the pial sheath are always situated within the conus, it is impossible that the anterior laminæ of the chorioid could have been present within the area of the conus at some previous period and have afterwards disappeared. The defect must have rather originally been the result of anomalous development, and not of disease.

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The changes in the posterior layers of the chorioid might be looked upon as atrophic if they were regarded independently. When, however, it is considered that in myopic eyes of normal length and shape there is an absence of any conditions which could have occasioned a sharply defined atrophy of a small part of the chorioid, and that in glaucomatous eyes of young subjects which have become greatly enlarged, and in which the originally normal chorioid has undergone a forcible distention, there is no circumscribed atrophy of the chorioid at the outer border of the papilla, and, further, that in the staphylomatous eyes of young myopes having a crescentic conus deficient development of the anterior layers of the chorioid is invariably present, it must be assumed that anomalies of texture in the posterior layers of the chorioid accompanying crescentic conus, like those in the anterior layers, are due to imperfect development. *The crescentic conus is, then, an agenesis of the optic nerve in consequence of an imperfect development of the chorioid, manifested in the malformation of that membrane at the lateral border of the chorioidal canal.*

The generally accepted opinion that the crescentic conus gradually becomes annular by the prolongation of its pointed extremities along the border of the papilla is erroneous. An annular conus cannot develop from a crescentic conus; neither can a crescentic one enlarge and become annular. The two forms of conus are, however, intimately related, for they are both anomalies in the development of the optic nerve, dependent upon imperfect development of the chorioid, or, sometimes, of both the chorioid and the sclera.

Every conus is, then, congenital, although in the eyes of adults who have been affected with myopia for years it may have become much larger than at birth. The conus, so small at first as to be scarcely distinguishable from a ring of normal connective tissue, increases in size with the eye.

Of the investigators who have studied the anatomy of the conus, von Jaeger alone recognized its dependence upon imperfect development. The conclusions of this observer are as follows: "When we consider the development of the human eye at different periods of fœtal life, and the existence and the manner of closure of the chorioidal fissure, the conus must be regarded not only in most cases as an indication of tissue-changes to appear at a later period of life, but also sometimes as the evidence of an anomaly in the closure of the chorioidal fissure."[1]

IV. The posterior staphyloma of Scarpa is most frequently situated in that part of the wall of the eyeball included in a circle described upon it, whose centre is at the outer edge of the optic nerve and its sheaths, and whose radius is equal to the distance between the optic nerve and the posterior end of the tendon of the inferior oblique muscle. As the diameter of the optic nerve is but a trifle less in length than the distance of the optic nerve from the posterior end of the tendon of the oblique muscle, the

[1] Loco citato, S. 69.

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staphyloma embraces only a narrow strip of the sclera on the inner side of the optic nerve, but a comparatively broad piece on the outer side. In the normal eye, the insertion of the posterior end of the tendon of the oblique muscle is about four millimetres from the optic nerve, while in an eye with posterior staphyloma this distance may amount to as much as seventeen millimetres. The more this part of the sclera is distended, the more attenuated it becomes. In eyes with large staphylomata the wall of the staphyloma may be as thin as note-paper.

In some myopic eyes a low circumscribed protuberance with a circular base is seen between the outer side of the optic nerve and the tendon of the inferior oblique muscle; in others, the surface of the sclera between the optic nerve and the inferior oblique muscle has undergone such marked enlargement that a conical elevation is formed on the outer side of the nerve; in others, again, the sclera immediately around the optic nerve is drawn into the wall of the staphyloma in such a manner that the nerve, instead of lying to one side of the staphyloma, forms its summit, the staphyloma then resembling the segment of a sphere, divided by a narrow furrow from the part of the eyeball not included in the staphyloma.

All these more or less sharply defined vesicular or conical protuberances in the wall of the eyeball are instances of genuine staphyloma. In the majority of cases, however, the thin portion of the wall merges gradually into the normally resistant part, the eyeball assuming the form of a cylinder with rounded ends, or that of an egg, with the optic nerve situated at its smaller extremity. The thinning of the sclera in such cases extends as far as the equator of the globe, and not infrequently beyond this region, to the neighborhood of the insertion of the rectus muscles. In eyes with posterior staphyloma the diameter of the base of the cornea, and the distance of the insertion of the rectus muscles from its border, are, however, never greater than normal. The length of eyeballs with the posterior staphyloma of Scarpa always exceeds that of the longest emmetropic eyeballs yet measured.

As there are many fully developed eyes with an axial length of only twenty-two millimetres, it may be assumed that eyes with posterior staphyloma exist whose length does not exceed twenty-five millimetres, although no record of such eyes has as yet been made. The axis of the shortest of the eyes with posterior staphyloma studied by the writer measured twenty-seven millimetres; that of the longest, thirty-two millimetres. The axes of the six staphylomatous eyes mentioned by Arlt varied between twenty-seven and four-tenths and thirty and seven-tenths millimetres in length. The shortest of the five eyeballs with posterior staphyloma measured by Donders was twenty-eight and five-tenths and the longest thirty-three millimetres in length. Von Jaeger found that, with the exception of one eyeball which was twenty-five and seven-tenths millimetres long, the axes of myopic eyes with posterior staphyloma measured from twenty-seven and seven-tenths to thirty-two millimetres.

As the thinning of the walls of eyes affected with posterior staphyloma

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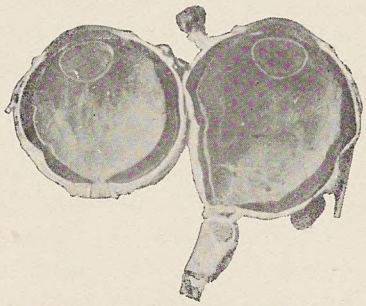
FIG. 7.

Oviform eyeball (left) with posterior staphyloma; axial length equalled thirty millimetres. Removed after death from a woman sixty-seven years of age. Beside it is the right eyeball of the same woman, which was emmetropic and had an axial length of twenty-four millimetres.

FIG. 8.



Pyriform eyeball (left) with posterior staphyloma. Length of axis equalled twenty-nine millimetres, horizontal diameter twenty-seven millimetres, vertical twenty-four millimetres. M. more than 20 D. Beside it is the right emmetropic eyeball of the same individual, the axial length of which was twenty-four and one-half millimetres.

FIG. 9.

Eyeballs of a woman sixty-seven years of age. M. 10 D. Length of axis equalled twenty-seven millimetres. In both eyes there was a sharply defined, slightly prominent posterior staphyloma with a circular base, situated between the lateral side of the tendon of the inferior oblique muscle.

FIG. 10.

The right eye of a woman fifty-six years of age, with a large posterior staphyloma. M. about 25 D. Length of axis equalled thirty millimetres, horizontal diameter twenty-four, vertical twenty-four and one-half millimetres. The tendons of the superior rectus muscle and the external rectus muscle, as well as those of the two oblique muscles, are represented in the drawing. The distance between the posterior end of the tendon of the inferior oblique muscle and the optic nerve is almost as great as the distance between the anterior end of the inferior oblique muscle and the cornea; that is to say, about seventeen to eighteen millimetres.

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usually extends far forward, their equatorial diameter is also, as a rule, greater than that of the normal eyeball. In twenty emmetropic eyes the writer found the shortest vertical diameter to be twenty-two millimetres, the longest twenty-four millimetres; the horizontal diameter measuring not less than twenty-two nor more than twenty-five millimetres. In twenty eyes with the posterior staphyloma of Scarpa he found sixteen whose vertical measurements were from twenty-four and five-tenths to twenty-seven millimetres, and nineteen whose transverse diameters measured from twenty-five to twenty-eight millimetres. In nine emmetropic eyes the maximum circumference at the equator was seventy-eight millimetres; among twenty eyes with posterior staphyloma he found eighteen whose equatorial measurement exceeded seventy-eight millimetres, the length varying between seventy-nine and eighty-nine millimetres.

A remarkable peculiarity of the sclera in eyes with posterior staphyloma is the entire, or almost entire, absence of the external fibre layers in the immediate vicinity of the optic entrance. In the normal eye the sclera is thickest just at the orbital side of the dural sheath, because at that point the outer layer of the sclera is about twice as thick as at the inner. The most anterior part of the intervaginal space of the normal eye lies, therefore, within the sclera. If the anterior plane reached by the summit of the intervaginal space be prolonged in the sclera, the prolongation in a normally constructed eye will lie between the inner and middle thirds of the sclera; in eyes with posterior staphyloma, however, it is situated upon the posterior surface of the sclera. The sclera on the orbital side of the dural sheath is then in these eyes no thicker than at the top of the intervaginal space, or but a trifle thicker; in other words, the sclera in the immediate vicinity of the optic nerve is deprived of its outer layers.

The dural sheath in the normal eye is pressed by the thick outer layers of the sclera against the inner sheath, so that the anterior extremity of the intervaginal space resembles a fissure. In eyes with posterior staphyloma, in which the outer layers of the sclera are absent, the dural sheath unites with the posterior surface of the inner layer of the sclera at a considerable distance from the surface of the optic nerve, the anterior extremity of the intervaginal space thereby acquiring abnormal width and shape. Von Jaeger was the first to describe and illustrate this anomaly of form and size.

After it had become customary to consider the presence of a conus as pathognomonic of posterior staphyloma, the latter anomaly was looked upon as the cause of typical myopia. In 1876, Arlt expressed himself as follows: "When we speak of myopia in the ordinary acceptation of the term, we now think, as a matter of course, of an elongation of the eyeball in a sagittal direction; in every case of myopia of this kind there exists a more or less clearly marked ectasia in the neighborhood of the posterior pole."[1]

[1] Ueber die Ursachen und die Entstehung der Kurzsichtigkeit, S. 2.

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It was taken for granted that in the eyes of myopic school-children the normally thick sclera had been subjected to a process of distention, and the only question was whether in such cases the sclera had a congenital predisposition to ectasia under the influence of eye-strain, or whether it might become ectatic in the eyes of any child from excessive eye-work.

Since all authors who have had opportunities for the anatomical examination of eyes with those degrees of myopia which are acquired at school unite in asserting that such eyes are, as a rule, free from posterior staphyloma, it will not be necessary to enumerate and criticise the hypotheses that are explanatory of the acquirement of posterior staphyloma by pupils of the higher schools, with which the literature of this subject is burdened.

It is well established that children who enter school with hypermetropic or emmetropic eyes do not acquire the degree of myopia which is almost invariably the concomitant of posterior staphyloma, and the eye-work required in the schools cannot, therefore, be regarded as the cause of the development of posterior staphyloma.

The degrees of myopia which are regularly associated with posterior staphyloma—M. 10 and over—are rare. Tscherning found among six hundred and twenty-seven myopes only fifty-one whose myopia exceeded 9 D. Of these six hundred and twenty-seven cases of myopia found by Tscherning among seven thousand five hundred and sixty-four men from eighteen to twenty-five years of age, thirteen occurred among two thousand three hundred and thirty-six individuals who were occupied more or less with near-work; thirty-eight among five thousand one hundred and eighty-seven individuals in whom this, as a rule, was not the case. Of the two thousand three hundred and thirty-six eye-workers, four hundred and twenty—*i.e.*, eighteen per cent.—were myopes; of the five thousand one hundred and eighty-seven laborers, two hundred and seven—*i.e.*, four per cent.—were myopes. In the light of these figures the marked influence of eye-work upon the development of myopia is very convincing. Of the four hundred and twenty myopic eye-workers, thirteen—*i.e.*, three per cent.—had myopia exceeding 9 D.; of the two hundred and seven myopic laborers, thirty-eight—*i.e.*, eighteen per cent.—had myopia exceeding 9 D. From these figures it is equally evident that eye-work is not concerned in the development of posterior staphyloma.

As early as 1861 Von Jaeger said that as many eyes with normal length of axis are found among those individuals whose occupation necessitates continued strain of accommodation as in opposite conditions, and that in that class of society which, as a general thing, is not accustomed to overtax its eyes by excessive accommodation, there are as many or even more individuals who are myopic in consequence of posterior staphyloma as in the remaining classes of society.[1]

In order to arrive at a clear understanding of the origin of posterior

[1] Loco citato, S. 28.

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staphyloma, two things must be understood: (*a*) the fact that the high degrees of myopia which are to be referred to this condition have, as a rule, been present from the earliest period at which the individual can give any account of his visual powers. Those suffering from myopia of more than 10 D. frequently assert that their myopia has increased, but they also generally state that they were never able to see at a distance. The writer does not know of an instance in which he could satisfy himself that an individual with myopia exceeding 10 D. had ever had normal vision in childhood and had acquired myopia at school, and that the myopia so acquired had developed from a low to a medium and finally to an extreme degree. General experience shows that the myopia acquired by eyes originally emmetropic or hypermetropic does not attain the high degree met with in posterior staphyloma, and that high degrees of myopia are not the more advanced stages of low degrees. It appears paradoxical that low degrees of myopia do not develop into higher ones, and that the high degrees should not have been preceded by lower errors. These propositions, however, express the actual state of the case. (*b*) It is certain that the eyes of children with high myopia show no symptoms of those diseases which lead to the enlargement of originally normal eyes, and that in such eyes no evidence of disease of the outer membranes or of increased intra-ocular pressure is present. Eyes which in youth become enlarged in consequence of increase of intra-ocular tension never acquire the form that characterizes eyes with posterior staphyloma. They suffer from glaucomatous excavation of the optic nerve, but never develop a conus. Eyes with posterior staphyloma, on the other hand, generally have a conus, but never show a glaucomatous cupping of the optic nerve.

The sclera of eyes with a myopia exceeding 10 D. is thinnest in the neighborhood of the optic nerve. Particularly is this so in the region between the outer border of the nerve and the posterior extremity of the tendon of the inferior oblique muscle. In these eyes there is never superficial enlargement of the portions of the wall of the eyeball anterior to the insertion of the rectus muscles. In eyes that have undergone glaucomatous distention the distention of the globe is never limited to the part of the sclera situated about the optic nerve, but enlargement of the cornea and of the scleral surface anterior to the recti is very frequent. Although such eyes often present uniform enlargement of their walls, and may have corneal, intercalary, ciliary, and equatorial staphylomata of various sizes and positions, they never have posterior staphylomata.

Eyes with posterior staphyloma in which the myopia exceeds 10 D. are especially liable to retino-chorioiditis and detachment of the retina; eyes that become enlarged under demonstrable increase of tension are not subject to either of these affections. Inflammation of the ocular membranes and increase of intra-ocular tension are not factors in the development of posterior staphyloma.

(*c*) Posterior staphyloma of Scarpa occurs almost exclusively in eyes

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presenting defective development of the chorioid and an anomalous form of the optic nerve (the conus) due to it. There is also generally an attendant malformation of the sclera, shown by an absence or rudimentary development of its outer layers in the vicinity of the optic nerve, with resulting anomalous insertion of the dural sheath and consequent abnormal size and shape of the intervaginal space.

The conus has been regarded as a consequence of scleral ectasia, and the stretching of the sclera due to the ectasia as the cause of the unusual insertion of the dural sheath.[1]

It is also asserted that the outer layers of the sclera become separated from the inner, and that the width of the intervaginal space is increased by the interval thus formed between the outer and inner layers of the sclera. The writer can state most emphatically, from an extensive series of examinations, that the enlargement of the intervaginal space in eyes with posterior staphyloma is never due to separation of the scleral lamellæ, and that the outer wall of the wide intervaginal space—*i.e.*, the one farthest removed from the optic nerve—is always covered by the arachnoidal sheath; that the abnormally large intervaginal space in myopic eyes is always surrounded by the two vaginal sheaths and the posterior surface of the inner layer of the sclera, just as in the normal eye, and never by the separated scleral laminæ. He has also demonstrated the existence of an excessive widening of the intervaginal space in an eye with myopia of 2 D. and axial length of twenty-five and five-tenths millimetres, in which there was no posterior staphyloma. If the abnormal width of the intervaginal space were caused by stretching of the sclera, it would be most marked on the side of the optic nerve corresponding to the summit of the staphyloma and the site of the crescentic conus. The contrary, however, is the rule, for the intervaginal space is much wider on the nasal than on the temporal side of the optic nerve. If the intervaginal space of eyes with high myopia had acquired its abnormal width from gradual distention of the sclera, the arachnoidal trabeculæ, distributed over a larger surface, would be correspondingly more attenuated at every level of the inner surface of the dural sheath than in the normal eye. As a matter of fact, the thickness of the arachnoidal trabeculæ in the wide intervaginal space of highly myopic eyes is much greater than in the normal intervaginal space. If the outer sheath had been forcibly separated from the inner it would be tense, its fasciculi would be stretched, and the shape of the intervaginal space would resemble a ninepin with its base situated at the sclera. Instead of this the sheath is relaxed, its fasciculi are wavy and have the appearance of being too long, while the form of the intervaginal space is variable and irregular.

Congenital anomalies in the development of the chorioid and sclera are, therefore, constant and essential features of eyes with posterior staphyloma. In view of the facts that posterior staphyloma is known to have

[1] See Arlt, Ueber die Ursachen und die Entstehung der Kurzsichtigkeit, S. 9 u. S. 30.

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existed in the early childhood of myopes of high degree, that no proof has as yet been furnished that the sclera of eyes with such a condition ever possessed normal thickness and shape, that its development is not preceded by any disease of the eye, that there is no increase of tension in staphylomatous eyes, and, finally, that inflammation and increase of intra-ocular tension do not cause posterior staphyloma, but a different form of bulbar enlargement, it is certainly warrantable to conclude that the condition is an anomaly of development in the eye, resulting from congenital peculiarities of texture in the parts of the chorioid and sclera surrounding the optic nerve.

It is a well-established fact that in cases of incomplete closure of the chorioidal fissure, in which a so-called coloboma of the chorioid in the lower part of the wall of the eye, or in the macular region, is seen with the ophthalmoscope, the part of the sclera that lies behind the coloboma of the chorioid is thinner than normal, has an unusually large superficial area, and is ectatic. Defective development in this instance is due to malformation in the ocular membranes. The posterior staphyloma of Scarpa is to be classed with such staphylomata. As the investments of these staphylomata are from the beginning abnormal in form and texture, and do not become thin and ectatic in consequence of some chance disease of an originally normal sclera, so the thinness and ectasia of the posterior staphyloma of Scarpa are due to malformation, and are not the consequence of disease of a normal sclera. We must, therefore, accept the opinion of von Jaeger, who concludes his observations upon posterior staphyloma with the remark that this condition must be regarded as an anomaly in the form of the eye, the shape assumed in any special case being dependent upon some accidental peculiarity of development.[1]

V. Posterior staphyloma is, then, a malformation, not a consequence of disease. It cannot, therefore, be acquired by eyes with normal membranes. The contrary opinion, which has given rise to the belief that thousands of entirely normal eyes acquire posterior staphyloma every year in the schools, is based upon the confusion of conus with posterior staphyloma, and upon the erroneous assumption that eyes with conus have a superadded posterior staphyloma. Every eye with the posterior staphyloma of Scarpa is myopic, but only a very small number of myopic eyes have this type of staphyloma.

As Herrnheiser and the writer have examined eleven eyes with myopia of from 2 D. to 6 D. without finding a posterior staphyloma, and as Arlt, Weiss, Stilling, and Krotoschin have examined nine eyes with from M. 3 to M. 6 in which no staphyloma was present, it may be regarded as almost demonstrated that posterior staphyloma is present only in eyes whose myopia exceeds 6 D. Von Jaeger alone mentions five eyes with myopia of from 1/24 D. to 1/7 D. that had posterior staphyloma, but these eyes

[1] Loco citato, S. 72.

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had such long axes that it is justifiable to question this author's estimate of the amount of the myopia.

As among twenty-one eyes whose myopia amounted to at least 10 D. the writer found only one which had neither conus nor staphyloma, but a normal sclera and an axial length of twenty-seven millimetres, and as staphylomata have been found by numerous investigators in many eyes with myopia of not less than 10 D., it is warrantable to assume that an eye with M. 10 or over is probably affected with posterior staphyloma. So few anatomical studies of the degrees of myopia between 6 D. and 10 D. have as yet been made that little is known of the relative frequency of the dependence of the myopia in these cases upon posterior staphyloma. The writer has examined but one eye with M. 7 and one with M. 8 anatomically. The former was twenty-seven millimetres long, and had a normal sclera and no conus. The latter was twenty-six millimetres long, had a conus, but did not show any posterior staphyloma. Von Jaeger describes an eye with a myopia of 1/6 D. and an axial length of thirty and three-tenths millimetres in which there was a posterior staphyloma, and another with myopia of 1/5 D. which had an axial length of thirty-one millimetres and possessed a posterior staphyloma. No anatomical examinations of eyes with M. 9 have as yet been reported.

If the writer could dismiss his doubts as to the correctness of von Jaeger's estimate of the degree of myopia present in the two eyes the respective axial lengths of which were thirty and three-tenths and thirty-one millimetres, he would say that one-half of the eyes having from 7 D. to 8 D. of myopia have posterior staphyloma. Anatomical data concerning myopia of this degree are as yet so scanty that it is best to defer judgment. It may be stated provisionally that the degrees of myopia intermediate between those that, as a rule, result from posterior staphyloma and those that are not usually attended by this anomaly sometimes have staphylomata and sometimes do not. To this category belong the myopias which exceed 6 D. but are less than 10 D.

If the various degrees of myopia are classified according to the frequency of the occurrence of macular chorioiditis, results which accord perfectly with those furnished by anatomical examination are arrived at. Among three thousand four hundred and ninety myopic eyes, Horstmann found two hundred and thirty with macular affections. In two thousand one hundred eyes, myopia from 1 D. to 6 D. was present; in eight hundred and eight eyes, myopia from 6 D. to 10 D.; and in five hundred and eighty-two eyes the degree of myopia was less than 10 D. In the first group, twenty-one (one-tenth of one per cent.) had macular affections; in the second, seventy-six (nine per cent.); and in the third, one hundred and thirty-three, or twenty-three per cent.[1] Among five thousand and thirty-nine myopic eyes, Schweizer found two hundred and sixty-five with macular disease, four

[1] Beiträge zur Myopiefrage, Charité-Annalen, Bd. v. S. 408.

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thousand and twenty-nine had from M. 1 to M. 6, four hundred and seventy-five had from M. 7 to M. 9, and five hundred and thirty-five had from M. 10 to M. 35. In the first group there were twenty eyes, or one-half of one per cent., with macular disease; in the second, twenty-four, or five per cent.; in the third, two hundred and twenty-one, or forty-one per cent.[1] Though in the myopias up to 6 D. retino-chorioiditis of the macular region is not more frequent than in non-myopic eyes, in the myopias exceeding 10 D. it is found to be twenty-three times, or even, according to Schweizer, eighty-six times, more frequent.

These facts constitute an urgent reason for classing the myopias up to 6 D. in one group, those exceeding 10 D. in a second, and those that are greater than 6 but less than 10 D. in a third. In the myopias of the last-mentioned grade, according to Horstmann, macular affections occur nine times, and, according to Schweizer, ten times, more frequently than in those of the first group.

The eyes of the first group are, as we have already shown, those which, as a rule, have no posterior staphyloma; the eyes of the second group are those in which, almost without exception, the anomaly is present.

The dangers which threaten myopic eyes are, then, associated with posterior staphyloma, a malformation which is as frequently found among the illiterate as among the educated and others whose eyes are subjected to the strain of close work. We are able to show only that a connection exists between retino-chorioiditis and posterior staphyloma: we cannot explain it. We can point out that the macula lutea lies in that part of the wall of the eyeball which in posterior staphyloma exhibits the greatest degree of textural change,—that is to say, in the part between the temporal edge of the optic nerve and the posterior end of the tendon of the inferior oblique muscle,—and, further, that the existence of other malformations also leads to early loss of the eye. Eyes with coloboma of the chorioid become blind from irido-cyclitis much more frequently than those that were originally normal; but the writer has found it impossible to ascertain any cause for this beyond the existence of a malformation.

The primarily emmetropic or hypermetropic eye is in no danger of becoming affected with retino-chorioiditis of the macula in consequence of acquired myopia. Only eyes with posterior staphyloma resulting from congenital malformation have, in addition to excessive myopia, an especial predisposition to that grave disorder.

[1] Ueber die deletären Folgen der Myopie, Archiv für Augenheilkunde, Bd. xxi. S. 399.

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DISEASES OF THE RETINA.

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I. THE NORMAL FUNDUS OCULI.

In the normal fundus the retina is invisible, its larger blood-vessels alone being seen. Its color varies from a light yellowish red (seen in albinos and the fairest Caucasians) to a dark chocolate brownish red (seen in the negrine races), and seems to be but slightly influenced by the visual purple. The want of pigment in the retina of albinos permits a view of the chorioidal vessels, and even at times of the sclera, which latter gives to the eye-ground a brilliant, somewhat tessellated, pinkish appearance. In the ordinary blonde the chorioidal vessels are only occasionally seen. Exceptionally the sclera is visible. In brunettes a brownish-red fundus is the rule. The same pigment tendency that pervades all the tissues of the brunette shows itself here in the heaping up of pigment in the intervascular spaces of the chorioid, giving a regular mosaic appearance to the eye-ground. In such fundi the retina, quite a distance from the papilla, is sometimes recognized as a delicate, misty-gray veil, which a novice might easily mistake for a chorioidal lesion. In the negrine or negroid races the eye-ground from the bulbar equator to the ora serrata presents a chocolate brownish red, while near the posterior pole of the globe it appears of almost a slaty gray. In children, especially in hypermetropic subjects, a peculiar satin-like sheen radiating from the papilla is frequently superadded, a condition which a beginner will not find it difficult to confound with diffuse retinitis or medullated nerve-fibres.

During mental or physical anguish the nerve head blanches, the arteries and veins become thinner, and the latter often appear as if broken. Just at death the papilla is blanched, the whole fundus suddenly pales, the arteries become narrow, and the veins empty centripetally, their blood-columns breaking until they look like strung beads. Half an hour after death the eye-ground is yellowish white, the papilla is chalky white, the arteries are

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invisible, and the veins are thin and almost empty. This picture is an infallible sign of general dissolution, and it is rather surprising that, though this fact was pointed out by Poncet[1] and Aldridge[2] fully twenty-five years ago, it has never been utilized.

The optic papilla or nerve head does not lie at the exact centre of the posterior pole of the eye. It is situated about one disk-diameter to the nasal side of the centre. It appears as a light pink disk, which is generally either round or slightly oval. In normal eyes it has well-defined edges. As the proportions of the disk are fairly constant, it is frequently employed in measuring or stating distances in the background of the eye. It is bounded by two rings. The inner one, which is white, is known as the scleral ring. The outer, which is rarely complete, is mostly represented by separate aggregations of pigment, especially at the sides, this black circlet being termed the chorioidal ring. These disk-outlines are usually less marked nasally, because in this situation a larger number of nerve-fibres leave the edge of the disk. This accounts also for the slightly redder tint of the nasal half of the papilla.

The nerve head lies at the same level as the rest of the background, and presents at its centre a funnel-shaped depression known as the *physiologic excavation*. This excavation varies in size. Sometimes it is very small, though usually it occupies the central third of the papilla. Exceptionally it may reach almost to the scleral ring. The whiteness of the excavation is due to the reflex from the underlying lamina cribrosa.

The macula lutea, or yellow spot, generally lies from one and a half to three disk-diameters to the temporal side of the disk, and about half a disk-diameter below the horizontal meridian. Usually it is a transverse oval in shape, with a slightly greater long diameter than that of the optic disk.

In the centre of this transverse oval the fovea centralis is seen. It is about two-tenths of a millimetre in width, and just at its centre the foveola fundi can be discerned. Through the ophthalmoscope the macula appears but slightly different from the surrounding retina, notwithstanding it is the region of the retina to which is delegated the function of direct vision and sharpest sight. The only thing that can be noticed with the ophthalmoscope is that the eye-ground in this position is a shade darker. With subdued illumination a skilled observer will note a crescentic or comma-like reflex at the fovea, and at times he will see a white line surrounding the macular region. These appearances do not indicate structural alterations. They are simply light-reflexes which disappear when the illumination is too intense, the pupil dilated, or the focus changed. The macula is devoid of any visible blood-vessels.

All the retinal blood is supplied by the central retinal artery, and leaves

[1] Archives générales de Médecine, 1870, p. 408.
[2] West Riding Lunatic Asylum Reports, 1871, vol. i.

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the retina by the central retinal vein. The retinal blood-vessels are terminal in type. Only around the papilla, where the arterial circle of the optic nerve lies, is there any communication between the vessels of the chorioid and the nerve (as Heinrich, Müller, Zinn, and particularly Leber have found). The central vessels enter the eye through the physiological excavation in the optic nerve, dividing on the disk into several branches. The first division is usually into the superior and inferior papillary artery and vein. The vein is generally primarily bifurcated at the surface of the disk, while not infrequently the artery is still undivided in that situation. The second division of the vessels takes place upon the papilla, two branches of each kind generally passing in a nasal direction and two in a temporal one. These are called (1) the superior and inferior nasal arteries, which, branching always dichotomously, supply the smaller inner half of the retina; (2) the superior and inferior temporal arteries, which, branching also dichotomously, are larger and sweep in a large arc around the macular region, supplying, if the nerve head be assumed as the centre of the blood-supply, the larger outer half of the retina; (3) the median artery, which is a single branch taking its origin from either the superior or the inferior nasal artery and running medially between them; and (4) the superior and inferior macular arteries, which usually spring independently from the papilla and reach out almost to the macula (usually there are two of these, occasionally there may be three, and rarely there is but one).

The veins generally correspond, in position, distribution, and name, to the arteries.

Anastomosis between the arteries, in their larger or finer ramifications, is seldom if ever seen, while inosculation between the veins sometimes occurs in the periphery of the eye-ground; practically, therefore, the only communication between the retinal blood-vessels is by way of the capillaries. The macula is supplied by a wreath of capillary loops situated about the fovea, although the fovea itself is without direct blood-supply. The bright white streaks seen in the course of the vessels, generally the arteries, are, as a rule, simply light-reflexes. (Jaeger.) At times the veins are seen to pulsate normally at the physiological excavation. If this pulsation is not present it can be easily induced by slight pressure on the eyeball with the finger. Arterial pulsation can also be produced in a similar manner, though it requires more pressure. Spontaneous arterial pulsation is always pathological.

II. SENILE RETINAL CHANGES (SENESCENTIA RETINÆ).

Senile changes in the retina vary from the simplest form, in which the retina gradually loses its almost transparent brilliancy, to those phases that are difficult to distinguish from the gravest chorioidal and retinal lesions.

In the simpler forms the retina appears veiled, especially about the disk. Sometimes dull gray points and molecular masses, giving the tissue

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a marbled appearance, are found.[1] In such retinæ Mauthner and von Stellwag have observed round, highly refractive bodies which exhibit a dull sheen. In proportion to age there is also found a reduction of both central and peripheral visual acuity.

A second form of senescentia retinæ is due to colloid excrescences of the lamina vitrea of the chorioid, as described by de Wecker,[2] Iwanoff,[3] and Leber.[4] These excrescences disorganize the retinal elements as they force their way through its tissue. Occasionally they loosen from the chorioid and wander into the retinal tissue, where they may form globules which protrude considerably above the level of the retina. The ora serrata is the sole seat of these lesions. As seen with the ophthalmoscope, they appear as numerous, whitish-yellow, round spots which are slightly raised above the eye-ground and are usually bordered with pigment. Their number increases and they are more closely packed as the periphery of the membrane is approached. Scattered among them are smaller black points, which in the original stage represent the alterations just mentioned, appearing at the time when the excrescence about to enter the retina pushes the pigmentary epithelial layer before it. In this condition central visual acuity may remain unaffected, and only the most painstaking perimetry can give a clue to the true state of the retina by the appearance of scotomatous areas. Though clinically of little importance, it is necessary for the clinician to differentiate this phase of senescentia retinæ from disseminate chorioiditis. The diagnosis turns on the parallactic movement and the character of the plaques. In the former the plaques are small, roundish, raised above the adjacent eye-ground, symmetrically arranged, and bordered with pigment. Moreover, they do not coalesce, and are found peripherally. In the latter the plaques are large, irregular, depressed below the plane of the adjoining retina, and irregularly arranged about the posterior pole of the eye. In older cases the pigment is heaped and scattered and the plaques are confluent. During an experience of thirty-five years the writer has seen numerous instances of senile retina, one striking example of which was complicated by synchysis scintillans (reported below). In another instance, in which microscopic sections of the eyes of a subject of this disorder were made by the writer, an examination of the sections confirmed in every way the findings of Iwanoff.

Retrogressive metamorphosis in the retina gives rise to a third form of senescentia retinæ, which is characterized by the presence of crystals of carbonate of lime or cholesterin. This variety may complicate the ones just mentioned, when lime or other products of a regressive type are deposited in the colloid excrescences, as shown in a unique case by Nagel,[5] which was

[1] Wedl, Atlas.
[2] Graefe und Saemisch, Handbuch der gesammten Augenheilkunde, Bd. iv. S. 609.
[3] Klinische Monatsblätter für Augenheilkunde, 1869.
[4] Graefe und Saemisch, Handbuch der gesammten Augenheilkunde, Bd. v.
[5] Klinische Monatsblätter für Augenheilkunde, 1875, S. 338.

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proved by microscopical examination. This author doubts whether cholesterin crystals are ever found in the retina. Instances (unproved) have also been reported by Coccius,[1] who has studied one which was seen in connection with disease of the retina. Von Graefe[2] gives an example associated with amotio retinæ. Pagenstecher[3] cites one of the primary variety, Mauthner[4] one with neuritis, and Tweedy[5] one with pigmentary retinitis. Waren Tay and Hutchinson[6] were the first in England to describe the condition. Nettleship[7] has also studied it. Hirschberg[8] was the first to call attention to the disease in his country, while Goldzieher[9] has furnished one of the most complete descriptions. The case that will be reported farther on (senescentia retinæ cum synchysis scintillans corporis vitrei) will maintain that crystals of cholesterin are present in this kind of senile retina. Although we do not possess sufficient data to decide definitely, it is not likely that they disturb vision, for they have been found in normal eyes: so that when lowered vision does occur it must be due to retinal degeneration.

The ophthalmoscopic picture in this third variety might be easily confounded with that seen in retinitis albuminurica. The lesions are principally grouped about the posterior pole of the eye, although the retinal periphery is frequently involved. Arranged around the macula (Leber, Hirschberg) can be seen numerous whitish-yellow spots, sometimes merging into larger spots or plaques, which at times finally assume a pale rose color. The papilla is at times pallid. Near the retinal periphery are usually found small pigment massings. The central visual acuity is much lowered, while eccentric vision may be fairly good.

There remains to be mentioned a fourth variation, in which numerous plaques, found in the variety just described, coalesce in the circumpapillary region, forming a whitish-yellow ring that is bordered with pigment about the disk, and presenting a picture which to a beginner is strikingly suggestive of the posterior staphyloma of Scarpa. This form presents normal visual acuity, though an enlargement of Mariotte's spot can be determined.

It is almost needless to say that in all these phases of senile retinal degeneration treatment is of no avail.

In closing this section I wish to describe in detail a case illustrative of the second and third varieties of senescentia retinæ above referred to.

It was a case combining the features of senescentia retinæ scintillans with those of synchysis scintillans corporis vitrei, and was so brilliant that

[1] Die Augenspiegel, S. 122.
[2] Archiv für Ophthalmologie, Bd. ii.
[3] Klinische Beobachtungen, Bd. ii.
[4] Lehrbuch der Ophthalmoscopie.
[5] The Lancet, 1873.
[6] Royal London Ophthalmic Hospital Reports, viii.
[7] The Student's Guide to Diseases of the Eye.
[8] Contributions to Practical Ophthalmology, vol. iii.
[9] Wiener medicinische Wochenschrift, 1887.

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it was kept under observation for three years at Professor Hasner's clinic in Prague. During the years 1862 to 1865 the case was examined by colleagues from all parts of the world. The patient was a beggar, seventy-one years old. He had vision equal to the counting of fingers at one metre's distance. When the patient fixed steadily, innumerable whitish-yellow, roundish plaques, bordered with pigment, and varying in size, could be seen at the posterior pole of the eye. Here and there they contained streaks or islets of pigment. Between the plaques lay innumerable pigment heapings, some of which were oval or biscuit-shaped, while others were irregular and of various sizes. The eye-ground, as a whole, presented an appearance not unlike that of a Turkish carpet. Scattered over the entire background were glittering golden points and dots, which could be easily recognized as crystals, and which I am firmly convinced were cholesterin. When the eye was moved quickly several times and then again fixed, there was seen a shower of minute golden flakes slowly rising and falling in the vitreous. These viewed against the background of the eye made a truly fascinating picture.

Plate I. shows the appearance of the opacities against the eye-ground without regard to their coloration.

CONGENITAL ANOMALIES OF THE RETINAL BLOOD-VESSELS.

III. CILIO-RETINAL BLOOD-VESSELS.

As long ago as the end of the last century, Zinn ("Oculi Humani Descriptio") observed that fine branches of the posterior ciliary arteries sometimes pierce the optic nerve of a normal eye, branch out in the nerve-sheaths, and communicate with branches of the central retinal artery, thus promoting an anastomosis between the ciliary and central retinal circulations. Leber[1] showed that the connection between the two systems might be established either by way of the circulus arteriosus nervi optici or by way of the chorioidal margin, a fact which Donders[2] had suspected. Heinrich Müller[3] was the first to observe a large anomalous vessel probably arising from the posterior ciliary arteries and entering the retina by way of the chorioidal margin. Nettleship has recorded a similar picture seen in a pathological eye.[4] Schnabel and Sachs[5] have questioned the existence of cilio-retinal blood-vessels, but Birnbacher's work has proved their presence beyond a doubt. In a normal eye he found a large branch given off from a ciliary body, running first towards the optic disk and then entering the retina around the edge of the chorioid.[6] Two years previously Birn-

[1] Graefe und Saemisch, Handbuch der gesammten Augenheilkunde, Bd. ii. S. 306.
[2] Archiv für Augenheilkunde, Bd. i. S. 88.
[3] Ibidem, Bd. iv. S. 10.
[4] Royal London Ophthalmic Hospital Reports, vol. ix. p. 162.
[5] Archiv für Augenheilkunde, Bd. xv. S. 13.
[6] Ibidem, S. 292.

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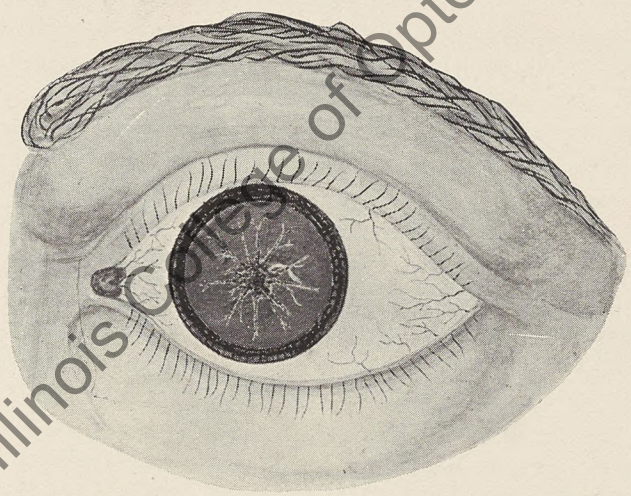
PLATE I.

Senile changes in the retina and the vitreous humor.

PLATE II.



Posterior polar capsular cataract (stellated form) with manifest canal of Cloquet.

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bacher[1] studied a similar condition in an eye that presented embolism of the central artery. The calibre of the cilio-retinal vessels remained unchanged even after the normal circulation had been re-established. In 1883 Benson exhibited a parallel case before the Ophthalmological Society at Heidelberg.

With the ophthalmoscope, a cilio-retinal vessel, if arterial, appears to take its origin just at the side of the disk or a little distance from it. If venous, it disappears at the same location. In 1871 Loring[2] described three cases, in two of which an enormous vessel disappeared at some distance from the disk. He thought that such veins either perforated the sclera independently or emptied into the chorioidal veins. While such fundi are not rare, care must be taken not to pronounce every vessel that takes its origin outside the disk to be a cilio-retinal vessel, for it sometimes happens that such vessels do not belong to the ciliary system, but branch from the central retinal vessels some distance behind the papilla, as in Jaeger's case.[3]

Vision is uninfluenced by the presence of cilio-retinal vessels. On the other hand, they may prove of inestimable value to their possessor, should he be so unfortunate as to be stricken with embolism of the central retinal artery, especially if, as often happens, the errant vessel runs in the direction of the macula.

IV. PERSISTENT HYALOID ARTERY AND MANIFEST CLOQUET'S CANAL (PERSISTENTIA ARTERIA HYALOIDEÆ ET CANALIS CLOQUETTI MANIFESTUS).

Persistent Hyaloid Artery.—According to embryologists, the primordial vitreous body owes its great vascularity to its mesodermal origin. The vasa hyaloidea primordialia are at first evenly distributed throughout the vitreous body, while at that time the retina is totally devoid of blood-vessels, a condition in which it remains throughout life in amphibians. As the vitreous body develops, these vasa primordialia attain their greatest development about its periphery and centre, while those of the intermediate zone atrophy. This atrophic process in the intermediate zone extends outward and inward, until finally there remain only central and peripheral vasa hyaloidea primordialia. The peripheral vessels gradually enter the retina, and thus form the retinal circulation. Of the central vessels but one medium-sized artery remains, which is called the hyaloid artery. This runs from the optic nerve head to the posterior pole of the crystalline lens through the canalis Cloquetti; in fact, it is a lymph-channel formed in the axis of the vitreous body. At the posterior pole of the lens the artery becomes the arteria capsularis postica, which, branching dichotomously, ramifies over the posterior surface of the lens and passes over its anterior surface into the membrana capsulo-pupillaris and the membrana pupillaris Wachendorfii, forming with these the tunica vasculosa lentis. According to

[1] Centralblatt für praktische Augenheilkunde, 1883.
[2] Archiv für Augenheilkunde, Bd. ii. S. 163.
[3] Beiträge zur Pathologie des Auges, Tafel I.

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most observers, the hyaloid artery with the tunica vasculosa lentis disappears in the middle of the fifth or the beginning of the sixth month of pregnancy. My own observations lead me to believe that this disappearance takes place frequently only a short time before birth. The obliteration begins at the posterior pole of the lens. In animals which are born blind the hyaloid artery and Wachendorf's membrane persist for eight or ten days after birth. I have often found the same thing in new-born children clinically, and have verified it by anatomical examination during my service at the Prague Lying-in Institute. In seven- or eight-months' viable children the condition is almost constant. Among full-term children persistent hyaloid artery is met with frequently in twins, and almost constantly in triplets or quadruplets. It is rarely seen in weaklings born at full term. So far as I have observed, these fœtal remnants disappear in from three to ten days after birth. In hoofed animals, especially cloven-footed ones, the origin of the canalis Cloquetti is often represented by a diaphanous cone attached to the papilla.

If the obliterated hyaloid artery is permanently retained in the human subject, we speak of it as a persistent hyaloid artery. If, however, the artery disappears and the canal alone can be shown clinically or anatomically, we speak of it as a visible canal of Cloquet. To these two conditions may be added the various processes and connective-tissue formations which result from intra- or extra-uterine inflammations.

Persistence of the hyaloid artery was first noted by Hannover,[1] and soon after by Arnold,[2] Stellwag,[3] and Wallmann.[4] It has also been seen by Finkbeiner[5] and by Meissner.[6]

Heinrich Müller has called attention to the almost constant presence of the hyaloid artery in the eye of the ox, urging ophthalmologists of wide observation to seek for similar conditions in the human eye. His appeal has been responded to by Saemisch and Zehender,[7] Liebreich,[8] and Toussaint.[9] In Toussaint's case the hyaloid artery was divided into three branches. Later Stör[10] and Laurence[11] have described cases which were complicated with posterior sclero-chorioiditis. Mooren's case,[12] in which the hyaloid artery took its origin from a branch of the central retinal artery, is unique. In 1893 Hirschberg[13] described a tube-like formation reaching from the papilla to the posterior pole of the lens, where it divided into several branches, in one of which blood could be seen. At the 1892 meeting of the American Ophthalmological Society, Mittendorf reported about

[1] Das Auge, 1852.
[2] Beiträge zur Anatomie.
[3] Zeitschrift des gesammten Wissenschaften, 1854.
[4] Ibidem, 1858.
[5] Zeitung für wissenschaftliche Zoölogie, Bd. vi.
[6] Zeitschrift für praktische Veterinair-Wissenschaften, Bd. i. S. 323.
[7] Klinische Monatsblätter für Augenheilkunde, Ss. 259, 260.
[8] Ibidem, S. 261.
[9] Ibidem, S. 350.
[10] Ibidem, S. 24.
[11] Ophthalmic Review, p. 173.
[12] Ophthalmologische Beobachtungen, 1867.
[13] Centralblatt für praktische Augenheilkunde, S. 360.

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forty cases of cataracta capsularis polaris postica lentis, which he believed were mostly derived from the hyaloid artery or the posterior lens-capsule.

My own experience embraces eight cases,—three out of a total of sixty thousand clinical, and the other five out of a total of one hundred and twenty thousand cases seen as Royal Provincial Oculist. The only bilateral case I have seen was associated with vitreous opacities and torpor retinæ. In this case vision equalled one-fortieth of normal. Incidentally, while examining the eyes of school-boys and students, I have observed persistent hyaloid artery three times in myopes and once in a hypermetrope. In like manner I have found a persistent hyaloid artery in the right eye of a girl who came for treatment of a cleft palate. I also have seen one case associated with posterior polar cataract and one with coloboma iridis.

Persistent hyaloid artery is not infrequently accompanied by other congenital arrests of development, such as anomalies of the central retinal vessels. Such cases have been reported by Liebreich:[1] for example, irideremia has been seen by Tukusch,[2] and coloboma of the optic nerve has been described by Beyer.[3] The condition has been noted with coloboma of the chorioid as reported by Remak,[4] and with posterior cortical cataract as seen by Carres y Arago, Reup, and Galezowski.[5] Glaucoma and retinitis pigmentosa[6] have also been brought to notice as complications of this anomaly. In only one reported instance has persistent hyaloid artery been observed in two generations of the same family, Pflüger[7] having found persistent hyaloid artery and visible canal of Cloquet in a mother and her child.

In 1868 Stilling[8] demonstrated the persistence in the normal eye of a hyaloid canal which under inflammatory conditions frequently became invisible: this was probably a manifest canal of Cloquet. Two years later Flarer found clinically a band-like, translucent formation traversing the vitreous body, with coincident persistent Wachendorf's membrane. Later de Wecker[9] saw two cases. In 1876 Manz demonstrated the condition anatomically.

In a clinical and private practice embracing fully one hundred and fifty thousand cases I have seen five classic instances of canalis Cloquetti manifestus, three of which, strangely enough, were complicated with retinitis pigmentosa. One of these is as follows. The patient was fifty-two years of age. In addition to the ordinary changes found in pigmentary

[1] Transactions of the Pathological Society, London, vol. xxii. p. 220.
[2] Dissertation, Strassburg, 1885.
[3] Prager medicinische Wochenschrift, Nr. 35.
[4] Centralblatt für praktische Augenheilkunde, 1885, S. 9.
[5] Recueil d'Ophtalmologie.
[6] Ulrich, Klinische Monatsblätter für Augenheilkunde, Bd. xx. S. 240.
[7] Bericht der Augenklinik, Bern, 1882.
[8] Archiv für Augenheilkunde, Bd. xiv. S. 260.
[9] Traité des maladies du fond de l'œil, p. 48; and Graefe und Saemisch, Handbuch der gesammten Augenheilkunde, Bd. iv. S. 702.

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degeneration of the retina, the right eye showed a star-shaped posterior polar cataract. In the left eye there was a dim disk-shaped area situated at the posterior pole of the lens. From this a wide manifest canalis Cloquetti reached backward by spiral depressions and terminated in a ball-shaped end which almost covered the whole of the papilla. Whether a persistent hyaloid artery ran through its interior could not be determined, as the canal was opaque. (See Plates II. and III.)

When the hyaloid artery is atrophied but not obliterated, it appears as a blackish-gray string stretched from the nerve head to the posterior pole of the lens. Sometimes it is arched, and, if the vitreous be fluid, it may float whenever the globe is moved. Occasionally two or three branches are given off near the lens or before reaching it. Rarely the string is ruptured in progressive myopia. In the axis of the string a black streak is usually seen, while the peripheral parts are grayish and translucent. In rare cases, like Zehender's, the artery may appear red near the posterior pole of the lens, an appearance which Zehender believes is due to invisible capillaries, although I cannot assent to this proposition. Liebreich's observation of a hyaloid vein said to have accompanied the persistent hyaloid artery is certainly unique, but is inexplicable.

With persistentia canalis Cloquetti we usually find in the physiological excavation a minute projection surrounded by a more or less pronounced cylindrical transparent tube stretched from the papilla to the posterior pole of the lens. Otherwise the tunics and media generally appear normal. Sometimes bizarre connective-tissue formations are seen. Again, there may be complications, such as chorioiditis disseminata or areolaris, retinitis pigmentosa, or posterior polar or capsular cataract. This latter condition may be sometimes found independent of persistent hyaloid artery; yet genetically it must, as has been shown by von Ammon,[1] be referred to it.

Refractive anomalies are found in most cases of persistentia canalis Cloquetti, thus leading to its accidental discovery. With correction of the refraction vision is generally found to be normal. If, however, the complication is cataractous, or of the nature of an inflammation of the vitreous, uvea, or retina, vision suffers in proportion to the extent, importance, and locality of the particular disturbance. At times other congenital defects, such as microphthalmos or coloboma, are found to coexist, in which event visual acuity is reduced to a minimum.

V. PERSISTENT ECCENTRIC HYALOID ARTERY (ARTERIA HYALOIDEA PERSISTENS ECCENTRICA).

The term *arteria hyaloidea persistens eccentrica* is applied to that condition in which the fœtal vessel takes its origin, not from the central retinal artery in the physiological excavation, but from one of the chief branches that is more or less distant from the papilla.

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[1] Klinische Darstellungen, Bd. iii. S. 57.

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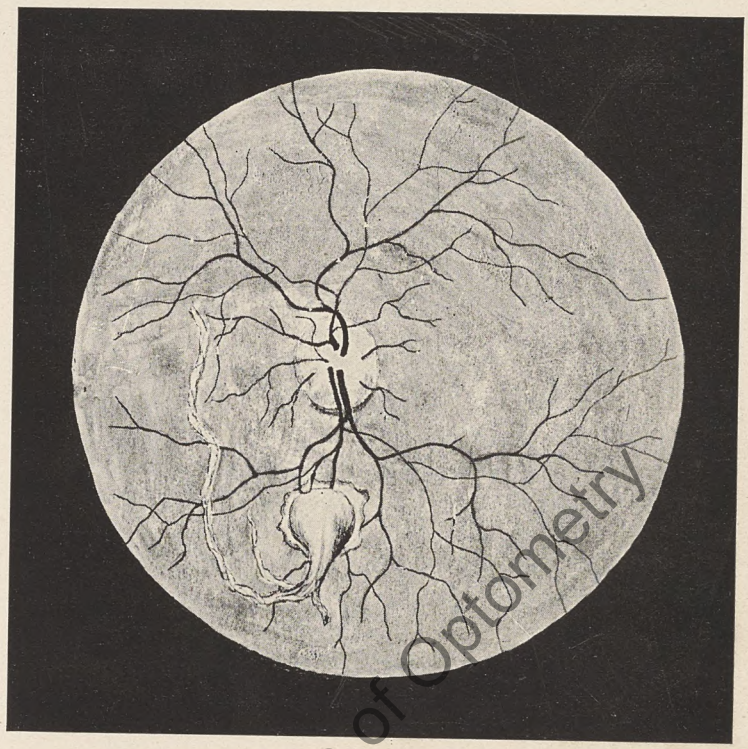
PLATE IV.



Ophthalmoscopic appearances of persistent eccentric hyaloid artery.

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Mooren's case and my own are the only ones, so far as I know, reported in ophthalmological literature. In Mooren's case[1] "the patient submitted unwillingly to an examination of a divergent strabismus. The vitreous was fluid, and there was iridodonesis. The ophthalmoscope showed a poorly developed optic nerve and retinal vessels. From a small arterial branch situated outward from the papilla sprang a grayish-blue band which traversed the vitreous body and reached almost to the lens."

My own case is as follows. A sixteen-year-old student came for relief from asthenopic symptoms occasioned by a low degree of ametropia. The ophthalmoscope revealed the following conditions. The media were clear. Refraction equalled —2.00 D. About one and a half disk-diameters down and in from the papilla could be seen a bluish-gray, somewhat translucent cone with a broad base, the diameter of which was about that of the papilla. From the apex of this cone sprang a short strand which passed to the right and there stopped. Farther forward a second, somewhat longer strand appeared, turned nasally, and ended abruptly. The cone continued forward as a bluish-gray string, extending as a slightly curved arch to the posterior lens-capsule, into which it was inserted with a slight enlargement a little below the posterior pole. From the papilla a main branch of the arteria centralis retinæ ran directly to the centre of the base of the cone, where it disappeared. A partially atrophic vein ran obliquely below the cone, while at its upper nasal margin could be seen a loop in which a fair-sized artery joined a vein of the same size. A black streak could be followed through the upper part of the cone, throughout the string, and even into the two branches. (Vide Plate IV.) With proper correction the patient's vision became normal, and only by the most careful perimetry could a central scotoma corresponding to the cone and Mariotte's spot be discovered. At my request the patient presented himself at my clinic and remained under observation there for some time.

The only explanation that I can offer in this case is that the black streak which ran through the main band and its branches represented an atrophied hyaloid artery of eccentric origin, and that the cone-shaped enlargement at the fundus was formed by the adventitial membranes. How this structure originated or developed it is difficult to conceive. Eversbusch's explanation of similar conditions accompanying persistent hyaloid artery would not apply to this case.

VI. PERSISTENT PSEUDO-HYALOID ARTERY (PSEUDO-ARTERIA HYALOIDEA PERSISTENS).

The occasional occurrence of post-embryonic formations not related to persistent hyaloid artery, and of certain membranous, string-like connective-tissue formations remaining after hemorrhages or chronic hyperplastic processes, especially when they start from the nerve head and run towards

[1] Ophthalmologische Beobachtungen, 1867.

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the lens, may easily mislead a beginner into the diagnosis of persistent hyaloid artery. Indeed, the false may so closely simulate the true condition that sometimes even an experienced ophthalmologist may err. It is not unlikely that many cases which have found their way into the literature of persistent hyaloid artery do not in reality belong there. Especially suspicious are those cases in which a hyaloid artery is described as complicated with progressive myopia, with chorioidal or retinal lesions, or with connective-tissue-like formations in the vitreous body. In high myopes flat, shell-like hemorrhages are not rarely seen in the macular and papillary regions, and it may happen in such cases that the blood escapes into pre-existing channels in the vitreous, disappearing later without a trace, or giving rise to the formation of a connective-tissue string, as in the cases of Unterharnscheidt[1] and Eversbusch.[2] The latter author saw in two different instances a large shell-like hemorrhage in one eye, while the fellow-eye presented a light gray band in the position of a persistent hyaloid artery which, to his mind, was undoubtedly the residuum of a former hemorrhage. In such cases the cylindrical lymph-space representing the canalis Cloquetti plays a part. In highly myopic eyes this space is somewhat enlarged in the area Martegiani, so much so that it was described by Lowe[3] as the so-called third chamber of the eye. When blood has entered this space a long time before the eye comes under observation, it may appear like a persistent hyaloid artery, but in recent cases the presence of a hemorrhage near the papilla will indicate the true condition. Stellwag records such a condition, in which a thin, sharply outlined column extended forward from the papilla and terminated at the posterior pole of the lens in a small disk-like extravasation of blood. A similar bilateral case fell under my own observation in the person of a teacher, who showed also the highest degree of myopia that I have ever seen.

While studying the hyperplastic inflammatory processes of the membranes of the eye, I have repeatedly seen real and apparent bands of connective tissue in the vitreous body, though they were not of sufficient importance to justify recording them. I have not critically separated all the published cases of arteria hyaloidea persistens from those of pseudo-arteria hyaloidea persistens. It will be sufficient if the foregoing statement serve to indicate the care that is needed in accurately diagnosing the manifest canal of Cloquet and persistent hyaloid artery.

VII. OTHER PRERETINAL VASCULAR STRUCTURES (VASA PRÆRETINALIA ADNATA VEL VASA HYALOIDEA PRIMORDIALIA PERSISTENTIA).

Under this heading will be considered those persisting fœtal vascular structures which, starting from the retina or the optic nerve, do not simulate

[1] Dissertation, Bonn, 1877.

[2] Mittheilung aus der Augenklinik, 1882, S. 60.

[3] Centralblatt für die medicinische Wissenschaft, 1878, S. 154.

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arteria hyaloidea persistens. This class should include those cases in which, along with grave malformations of the eyeball, especially coloboma oculi, separate blood-vessels or convolutions of blood-vessels persist.

The only instances of the condition of which we are now treating that I have found recorded have been by Nicati,[1] Rieban,[2] Little,[3] Czermak,[4] Hirschberg,[5] and Szili,[6] all of whom found as remains of primordial blood-vessels of the vitreous body several more or less complicated loops or arcades which sprang from retinal arteries and entered the vitreous body. The anomaly is exceedingly rare. Eyes exhibiting it are usually hypermetropic and possess good visual acuity.

ACQUIRED ANOMALIES OF THE RETINAL BLOOD-VESSELS.

VIII. NEWLY FORMED PRERETINAL BLOOD-VESSELS (VASA PRÆRETINALIA NEOPLASTICA).

It is no uncommon occurrence to find blood-vessels running from the retinal blood-vessels into newly formed connective tissue or membranous structures in the retina or the vitreous body, as, for instance, in retinitis proliferans and hyalitis proliferans. These new vessels are termed vasa præretinalia neoplastica, as distinguished from the primordial vessels. Although most frequently post-inflammatory in origin, they subsequently maintain a certain independence and enter the but slightly disturbed vitreous humor.

Coccius[7] first reported this anomaly. The adventitious vessels sprang from the papilla, projected far out into the vitreous, and floated slowly about when the eye was moved. Soon after, Jaeger[8] reported a case in which the development of the new vessels took place while the eye was under observation. Later Krause met with the anomaly[9] in a case of chorio-retinitis circumscripta. Nettleship records two cases, one complicated with syphilis, the other with diabetes.[10] Hirschberg has described one[11] in the eye of an old lady. He observed a flat, goblet-shaped, diaphanous formation situated in front of the papilla. It was traversed by numerously branched blood-vessels, underneath which the normal retinal vessels could be seen. The vitreous humor was transparent. The blood-vessels in the goblet were so regularly arranged that Hirschberg at first considered the

[1] Recueil de médecine vétérinaire et comparée, 1883.
[2] Charité-Annalen, 1876, p. 618.
[3] Transactions of the American Ophthalmological Society, 1881.
[4] Centralblatt für praktische Augenheilkunde, 1883, S. 239.
[5] Ibidem, 1883, S. 293, and 1885, S. 236.
[6] Centralblatt für praktische Augenheilkunde, 1885, S. 236.
[7] Ueber Glaucom, 1859.
[8] Beiträge zur Pathologie des Auges, 1871, S. 119, Plate XVI.
[9] Centralblatt für praktische Augenheilkunde, Bd. v. S. 48.
[10] Transactions of the Ophthalmological Society of the United Kingdom, 1884, p. 150, and 1888, p. 159.
[11] Centralblatt für praktische Augenheilkunde, 1890, S. 266.

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formation to be vasa hyaloidea primordialia persistentia. He also describes two other cases, one of which was associated with syphilis and the other with arterio-sclerosis.[1]

The ophthalmoscopic picture is characteristic. Slender blood-vessels can be seen to leave the papilla and penetrate for some distance into a transparent vitreous, in which place they often form delicate congeries. Accurate observation is sometimes difficult, from the fact that these formations lie in different planes and require separate focussing.

As to etiology, it might be said that these vessels are seen largely in old people, and that in almost every instance previous retinal or vitreous hemorrhages can be proved. Syphilis, diabetes, and arterio-sclerosis are to be viewed as distinct etiological factors. The disturbance of visual acuity is in direct proportion to the extent of the new formation. As to therapy, mercury is to be mentioned as having effected improvement after energetic application in some cases.

IX. RETINAL ANEURISMS (ANEURISMATA RETINALIA).

Retinal aneurisms are of very rare occurrence. They may be arranged in four groups: (1) aneurismata simplicia vera; (2) aneurismata miliaria; (3) aneurismata anastomotica miliaria multipla (tumores vasculosi arteriales miliares); (4) aneurismata arterio-venosa spuria retinalia.

The type *aneurismata simplicia vera* refers to those dilatations which occur singly on but one retinal artery. In rare cases they may attain quite large dimensions. In this class belong a number of observations of older date in which the condition was found post mortem, as, for example, those seen by the elder Graefe,[2] Schmidler,[3] and Scullet.[4] In all these the enlargement occurred on the central artery. The first ophthalmoscopic finding of a retinal aneurism was described by Sous[5] as a red ovoid tumor overlying the papilla and exhibiting a distinct pulsation. The other arteries were thinned, while the veins were dilated. Galezowski has seen a pulsating sacciform aneurism close to a retinal artery,[6] while Story and Benson's case[7] showed an aneurism at the end of an artery. In 1887 Schmall[8] found a fusiform aneurism in the eye of a woman with a cardiac lesion. Similar aneurisms are spoken of by Uhthoff,[9] Rampoldi,[10] and Raehlmann.[11]

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[1] Centralblatt für praktische Augenheilkunde, 1889, Ss. 9, 270.

[2] Angiectasia, 1808, p. 33.

[3] Dictionnaire des sciences médicales, 1817, t. xxxi. p. 20.

[4] Quoted by Demours, Traité des maladies des yeux, 1818, t. i. p. 108.

[5] Annales d'Oculistique, 1865, t. liii. p. 241.

[6] Transactions of the Ophthalmological Congress, London, 1872.

[7] Transactions of the Ophthalmological Society of the United Kingdom, 1884.

[8] Archiv für Augenheilkunde, Bd. xxiv. S. 65.

[9] Bericht über die fünfzehnte Versammlung der Heidelberger ophthalmologischen Gesellschaft.

[10] Annali di Ottalmologia, 1889.

[11] Zeitschrift für klinische Medicin, Bd. xvi., and Klinische medicinische Blätter, 1889, S. 203.

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The type *aneurismata miliaria* refers to dilatations of miliary dimensions occurring in great numbers on the smaller retinal arteries, as found post mortem by Liouville associated with miliary cerebral aneurisms in a woman aged eighty-four, the subject of atheroma. With Charcot he described a similar case. A third one mentioned by him was seen by Boucheron and Magnan. In 1876 Galezowski saw the condition in a pregnant woman who had sustained a slight contusion of the eye, and in the same year Lefort demonstrated the anomaly before the Medical Congress at Bordeaux. Bouchut depicts a case in his "Atlas d'Ophthalmoscopie Médicale et Cérébroscopie," observed in a paralytic. Litten,[1] Poncet,[2] Mackenzie,[3] and Benson[4] have also contributed to the literature of the subject.

Aneurismata anastomotica miliaria multipla.—The example of this condition reported by H. Pagenstecher is probably unique. He found it in an eye with glaucomatous degeneration enucleated upon account of pain.[5] Why this case has since been erroneously cited by Leber[6] and others as an instance of varicosity of the retinal veins is inconceivable. We quote the author verbatim: "The first case was one of small multiple tumors in the retina. . . . Most of them were of the character of true vascular tumors,—that is, they consisted of a large number of blood-vessels,—loops which were held together by connective tissue. These formations usually sprang from arteries, which latter were greatly thickened and sclerosed. The walls of the veins, too, were considerably thicker than normal; but I was unable to find with certainty that any such formations sprang from the veins." These unequivocal statements of Pagenstecher leave no doubt that in this case we have had to do with multiple miliary aneurisms, which might be looked upon as miliary angiomata, but never as varicosities of the veins.

The type *aneurismata arterio-venosa spuria retinalia* has reference to dilatations usually occurring after traumatisms, in consequence of which direct communication between larger arteries and veins is produced. Certain non-traumatic cases generally classed with these are probably of congenital origin. Magnus[7] has seen a case, in a nurse aged twenty-four who had suffered a contusion of the eye. The second was studied by Fuchs,[8] in a young man who had sustained an injury to the eye and in whom the tumor pulsated on pressure. The third was a non-traumatic case, seen by Schleich,[9] in an eight-year-old boy, presenting an arterio-venous aneurism

[1] Berliner klinische Wochenschrift, 1881.

[2] Gazette des Hôpitaux, 1876.

[3] Transactions of the Ophthalmological Society of the United Kingdom, 1882.

[4] The Ophthalmic Review, 1883.

[5] Klinische Monatsblätter für Augenheilkunde, 1871, S. 425.

[6] Graefe und Saemisch, Handbuch der gesammten Augenheilkunde, S. 526.

[7] Archiv für pathologische Anatomie und Physiologie und für klinische Medicin, Bd. lx. S. 4, Tafel III.

[8] Archiv für Augenheilkunde, Bd. xi. S. 440, Tafel VII.

[9] Mittheilungen der Ophthalmologisches Klinik, S. 202, Tafel IV.

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in the macular region, several anastomoses between the arteries and the veins, an aneurisma simplex verum, and a varix on the papilla.

The ophthalmoscopic picture in all varieties of aneurisms is so striking as to need no comment. In simple cases vision is undisturbed; with larger aneurisms there may be a central scotoma; while in the graver conditions vision is likely to be seriously compromised. Etiologically, it need hardly be stated that local disproportion between the arterial pressure and the arterial resistance leads to aneurismal formation. Treatment is unavailing.

X. DILATATION OF THE RETINAL VEINS (PHLEBECTASIA RETINÆ).

While fulness and tortuosity of the retinal veins are often seen, true phlebectasiæ are rare indeed. We differentiate between phlebectasia cylindrica, phlebectasia fusiformis, phlebectasia cirsoidea or serpentina, and phlebectasia sacciformis or varicosa.

Under the name of telangiectasiæ retinæ, Schirmer[1] has reported a case of common cylindrical phlebectasia in an individual who exhibited telangiectasiæ in other regions. Jacobi has observed three cases of cirsoid phlebectasiæ.[2] Hirschberg,[3] Michaelson,[4] and Schleich[5] have also contributed to our knowledge of the subject.

MULTIPLE SEED-LIKE BEADED DILATATION OF THE VEINS OF THE RETINA AND BULBAR CONJUNCTIVA (BOTH EYES)—PHLEBECTASIA MULTIPLEX MILIARIS MONILIFORMIS RETINÆ ET CONJUNCTIVÆ BULBI (OCULI UTRIUSQUE).

This name I have given to a rare anomaly, a solitary instance of which has fallen under my own observation. The ophthalmoscopic appearance is shown on Plate V., while the conjunctival condition is exhibited on Plate VI.

The case was that of a sixteen-year-old girl whose eyes presented a striking picture. The conjunctival and scleral veins were full, and exhibited innumerable rosary-like enlargements lying close together. (Plate VI.) In other respects the conjunctiva was normal. The retinal veins showed the same type of beaded enlargements in both fundi, which were otherwise normal. Vision equalled 5/5. The patient was pallid, and had not menstruated for two months. Diluted Cologne water, as a spray, was ordered to be used locally upon the conjunctival membrane, and iron and dietetic measures were employed internally. The conjunctival and retinal conditions subsided with the reappearance of menstruation. Six months later the phlebectasiæ conjunctivæ et retinæ were reproduced under

[1] Archiv für Ophthalmologie, Bd. vii. S. 119.
[2] Klinische Monatsblätter für Augenheilkunde, Bd. xii. S. 255.
[3] Beiträge zur Augenheilkunde, Bd. iii. S. 38.
[4] Centralblatt für praktische Augenheilkunde, Bd. xiii. S. 106.
[5] Loco citato.

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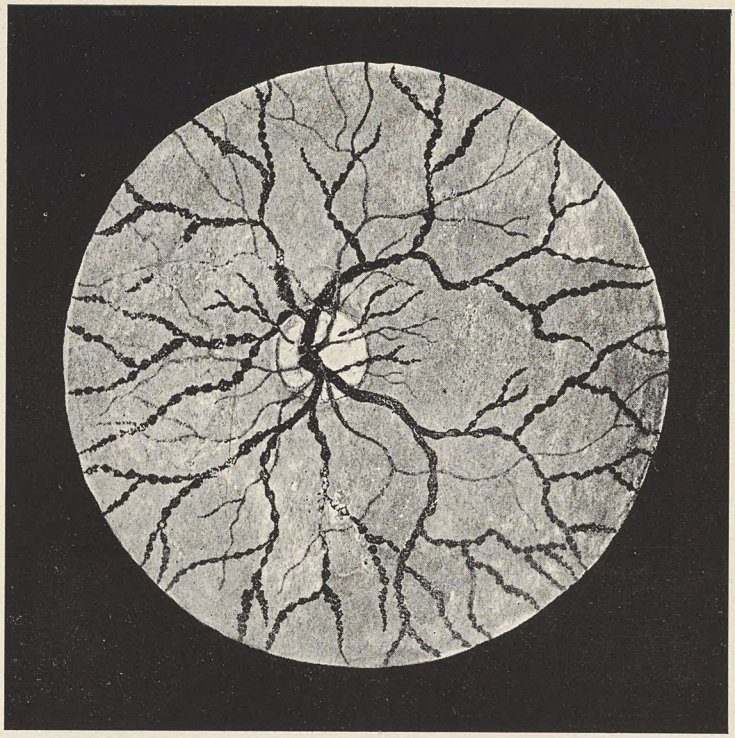
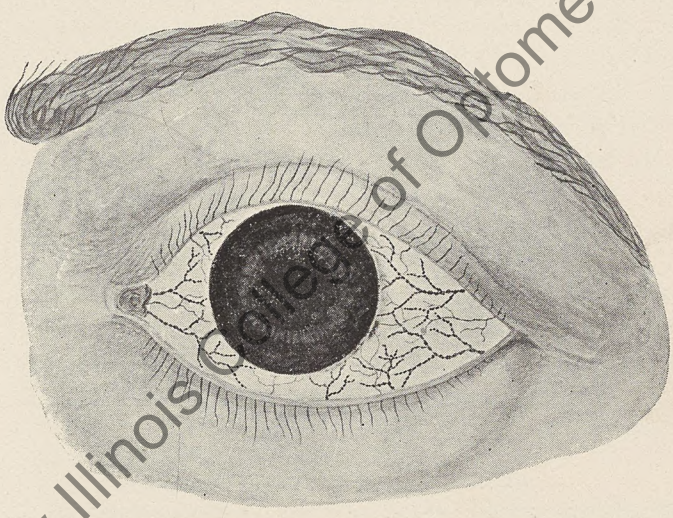
PLATE V.



Ophthalmoscopic appearance of phlebectasia of the retina.

PLATE VI.



Bead-like dilatation of the veins of the bulbar conjunctiva.

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the same conditions, to disappear again without leaving a trace after menstruation was re-established. Since then the patient has enjoyed perfect health.

Liebreich's case (Atlas, Plate IX., Fig. 1) simulates somewhat the retinal features seen in the above case, while Leber's case[1] resembles it in the conjunctival features, except that the ectasia was simple and uniform.

In the formation of cylindrical and cirsoid phlebectasiæ a simple obstacle to the flow of the venous blood is a sufficient cause. In the fusiform and sacciform varieties, affections of the vessel-walls, especially atrophy of the muscular and elastic elements, play a part. The phenomena in my own case I attribute to perverted vaso-motor influences.

XI. PERIVASCULITIS OF THE RETINAL VESSELS (PERIVASCULITIS VASORUM RETINALIUM).

This is an affection of the adventitia of the blood-vessels, occurring generally only as a symptom in different forms of retinitis, yet it may infrequently appear as a more or less independent disease, meriting, therefore, a separate description. For instance, Nagel[2] describes the retinal arteries as white bands in the eyes of a young man who, despite good central vision, showed defects in the visual fields. In Mauthner's case the arteries appeared as white bands, while the veins showed broad white borders.[3] De Wecker[4] and Jacobson[5] have described similar cases. There is no mistaking such a fundus. The arteries and their smaller branches (sometimes all the vessels) appear white or yellowish white. At times white-bordered veins will be found with the white arteries, and again the smaller vessels will be white while the larger branches will exhibit only a white border. The hypertrophy of the adventitia sometimes brings into view very small branches which under normal circumstances could not be seen ophthalmoscopically, thus giving rise to white dendritic figures in portions of the retina in which we are unaccustomed to find any blood-vessels. The visual disturbance is insignificant, inasmuch as the lumen of the blood-vessels generally remains intact. What visual impairment there may be is generally due to the primary affection or to complications.

The pathological conditions have been well studied by a number of authors.

While the tunica intima and tunica media are intact, the adventitia and the perivascular lymph-spaces are infiltrated with round cells, which may subsequently penetrate the vessel-walls, leading to secondary thrombosis or hemorrhages. The treatment is that of the primary condition.

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[1] Archiv für Ophthalmologie, Bd. xxvi. S. 191.

[2] Klinische Monatsblätter für Augenheilkunde, 1864, S. 394.

[3] Lehrbuch der Ophthalmologie, 1868,—depicted by Jaeger in his Atlas, Plate XVI., Fig. 75.

[4] Études Ophthalmologiques, t. ii. p. 224*b*.

[5] Archiv für Ophthalmologie, Bd. x. S. 58.

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XII. ARTERIO-SCLEROSIS OF THE RETINA (ARTERIO-SCLEROSIS RETINÆ).

This condition is often secondary to general senescence and arterio-sclerosis, and indeed may point to a similar condition in the cerebral arteries. Raehlmann's investigation of ninety cases[1] and Hirschberg's study of fifty[2] constitute the main contributions to our knowledge of this subject.

The eye-ground varies according as the case is recent or chronic. In most instances we find tortuosity and attenuation of the vessels, with the formation of white streaks along their margins. Arterial pulsation is not infrequent. In old and grave cases the larger vessels become white bands, the smaller ones disappearing, while at times aneurismal dilatation of the vessels may be seen. In early degeneration of the vessel-walls and obliteration of their lumen the picture changes, inasmuch as these conditions are frequently accompanied by multiple hemorrhages. Disturbances of function vary from slight reduction of visual acuity to contraction of the fields, torpor retinæ, and even sudden binocular blindness.[3]

The changes in the retinal blood-vessels consist in connective-tissue formations, complicated with degenerative processes affecting particularly the intima, and producing thick, rigid vessel-walls. The media is thinned, and sometimes shows hyaline degeneration, while the adventitia is thickened. The smaller vessels exhibit proportionately greater changes than the larger ones. The walls of the veins are sometimes, though rarely, uniformly thickened.

As stated by Thoma,[4] the early recognition of arterio-sclerosis retinæ is of great importance. Once found, great care must be exercised to prevent the development of the graver phases of the affection, by a well-ordered life,—diet, regimen, abstinence from severe mental or physical tasks, and avoidance of anything that may bring about increase of the intra-vascular pressure. This is as far as prophylaxis or therapy can go.

XIII. ANGIOEREMIA OF THE RETINA WITH ATROPHY OF THE RETINAL VESSELS (ANGIOEREMIA RETINÆ VEL ATROPHIA VASORUM RETINÆ).

Atrophy of the retinal blood-vessels is sometimes found after the type of inflammatory processes that lead to atrophy of the retina or the optic nerve. In such cases they appear like white thread-like streaks, which are apparently lost long before they reach the periphery. In advanced atrophy the vessels may be so shrunken as to seem wanting. Von Graefe speaks of a lack of retinal vessels[5] and of suspected absence of the whole retina,

[1] Zeitschrift der klinische Medicin, 1890, Nrs. 5 und 6, and Fortschritte der Medicin, 1889, Nr. 24.

[2] Centralblatt für praktische Augenheilkunde, 1890.

[3] Raehlmann, loco citato.

[4] Loco citato.

[5] Archiv für Ophthalmologie, Bd. I. S. 403.

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which were probably of congenital origin. Mooren refers to similar anomalous states.[1] In such conditions (for instance, in the ischæmia of embolism) the larger vessels appear like white bands, the smaller ones like threads. They differ from the structurally changed vessels of perivasculitis in the facts that they are still carriers of blood and that the smallest branches remain visible.

XIV. ANÆMIA AND ISCHÆMIA OF THE RETINA; SPASM AND EPILEPSY OF THE RETINAL VESSELS (ANÆMIA ET ISCHÆMIA RETINÆ; SPASMUS ET EPILEPSIA VASORUM RETINALIUM).

Any general anæmia, of whatsoever origin, is likely to induce retinal anæmia, which later finds its expression in constricted arteries, dark veins, and at times spontaneous arterial pulsation. According to von Graefe,[2] the condition has been observed in the terminal phases of cholera. Notwithstanding the diminished circulation, there is little, if any, impairment of vision in this simple variety of anæmia, and any treatment must have reference to the primary disorder only. However, in what is known as acute anæmia of the retina secondary to extensive hemorrhage, permanent amblyopia, or even total amaurosis, may ensue in from three to ten days. Illustrative cases have been recorded by fully fifteen different authors, every one of which was induced by great loss of blood. Paradoxical as it may seem, later it will be shown that hyperæmia instead of anæmia retinæ may appear in these cases. The fundus-picture is much the same as in simple anæmia,—that is, thin arteries, dark veins, and occasional spontaneous arterial pulsation, to which, in time, optic atrophy is usually superadded. The prognosis is gloomy. The therapy embraces roborant measures (iron, quinine, nourishing diet), resorbents (iodides), and incitants (strychnine hypodermically). Paracentesis of the anterior chamber and iridectomy have been unsuccessfully employed.

Ischæmia of the retina is the name given to the most profound retinal anæmia. It is seen as a symptom in embolism of the central artery, and in compression of that artery by disease or neoplasm. However, we shall speak here of those rare instances in which ischæmia occurs as a more or less independent affection. Cases have been reported by Alfred Graefe,[3] Heddeus,[4] Rothmund,[5] Secondi,[6] and Knapp,[7] in which the fundus arteries were found extremely thin, almost empty, and barely visible, the veins being broad and dark, and the papilla pallid. Notwithstanding there is grave disturbance of function, amounting to sudden total blindness, in such cases, the prognosis, judging from the above cases, in which vision returned to

[1] Ophthalmiatrische Beobachtungen, 1867, S. 260.
[2] Archiv für Ophthalmologie, Bd. xii. S. 198.
[3] Ibidem, Bd. viii. S. 143.
[4] Klinische Monatsblätter für praktische Augenheilkunde, 1865.
[5] Ibidem, 1866.
[6] Caso di Amaurose per Ischemiam, Turin, 1866.
[7] Transactions of the American Ophthalmological Society, 1880.

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normal in nearly every one reported, is hopeful. Roborants and paracentesis constitute the treatment.

Spasm of the retinal arteries, rarer even than ischæmia retinæ, sometimes produces the latter condition. It manifests itself in sudden and briefly temporary loss of vision. Hughlings Jackson [1] observed this condition in a woman who was blind for only five minutes. Zehender has seen a case [2] in which the blindness lasted seven minutes. Mauthner [3] and Zehender [4] state that Hornberger, of New York, studied the same symptom in his own person. No report of an ophthalmoscopic examination, however, is to be found.

Epilepsy of the retina is a term applied by Hughlings Jackson to a similar condition of arterial spasm which occurs during epileptic attacks. He has seen the retinal blood-vessels suddenly disappear while the fundus was being examined ophthalmoscopically at the time of the seizure.

XV. HYPERÆMIA OF THE RETINA (HYPERÆMIA RETINÆ).

Hyperæmia of the retina is characterized by increased calibre and tortuosity of its vessels, especially the arteries. These symptoms may be due to an absolutely or a relatively increased intra-vascular pressure, to alteration of the vascular elements, to pathological change in the vessel-walls, to changes in the intra-ocular tension, to affections of the nerves that regulate blood-pressure (especially to irritation of the sympathetic), to general plethora, and to intra-ocular or extra-ocular obstacles to venous outflow.

In arterial hyperæmia we find the optic papilla more or less red, but well defined. This redness is due to fulness of smaller arterial branches, ordinarily invisible, a similar condition being noticeable in the small vessels, this reaching almost to the fovea centralis. The arteries may be tortuous in the antero-posterior plane, rising in places, and again dipping into the outer retinal layers, causing them to appear at one part darker and at another lighter red. Spontaneous pulsation is frequently seen. Arterial hyperæmia is a concomitant expression of plethora; of eye-strain, whether due to muscular or accommodative asthenopia, or to working with poor illumination or on small glittering objects; of inflammatory processes in neighboring organs; of chorioiditis; of different forms of iritis; sometimes of deep-seated keratitis; of irritant applications to the conjunctiva (de Wecker); of the initial stage of retinitis; of diverse diseases of the heart and blood-vessels; of Basedow's disease; and of neurasthenia.

In 1876 E. Jaeger declared that general anæmia did not underlie anæmia of the retina as often as had been thought,[5] and in 1885 Raehl-

[1] Royal London Ophthalmic Hospital Reports, vol. iv. p. 15.
[2] Handbuch, S. 260.
[3] Loco citato.
[4] Loco citato.
[5] Ergebnisse der Untersuchungen der Ophthalmologie, 1876, S. 88.

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mann called attention to the seeming paradox of retinal hyperæmia associated with general anæmia dependent upon epistaxis, hæmoptysis, hæmatemesis, enterorrhagia, and metrorrhagia; he even saw retinal hyperæmia associated with chlorosis and cachexia. Strange to say, the veins appear light red and translucent.

Raehlmann found hyperæmia retinæ in sixty per cent. of his cases of general anæmia. He attributes these symptoms to a deficiency of corpuscular elements in the blood, which lessens the ordinary intra-vascular friction and thus permits an increase of blood-pressure in the peripheral blood-vessels. These cases are therefore to be viewed as oligocythæmiæ or hydræmiæ rather than as true anæmiæ. According to Friedreichson,[1] the quantity of hæmoglobin is reduced without often affecting the relative number of red blood-corpuscles.

Venous hyperæmia is characterized by suffusion of the papilla, coursing over which may be seen radiating vessels that are ordinarily invisible. Dilatation, tortuosity, and dark coloring of the veins are also present. The arteries are normal. If there be an additional obstruction of the arteries, these vessels will be even thinner and straighter than normal. In venous hyperæmia the obstruction may reside in the eye itself, as in glaucoma and some forms of retinitis. Again, it may be situated in the optic nerve or its sheaths, in the orbit, or even in the cranial cavity, in which case choked disk soon supervenes. Venous hyperæmia may also be seen in connection with meningeal congestion, with stasis or thrombosis in the cavernous sinus, with intra-cranial tumors, and with any obstruction found in the general venous circulation.

Hyperæmia of the retina is generally secondary to many ocular or general affections.

Treatment must necessarily have reference to the primary factor. The correction of any ametropia and the regulation of ocular work are always necessary. Cold compresses or eye-douches are sometimes requisite, and, if the trouble proceed from too intense illumination, smoked glasses will be of benefit. Occasionally cathartic waters are indicated.

Accepting Liebreich's idea, those conditions of exaggerated hyperæmia retinæ (especially venous) which are particularly connected with congenital heart-lesions, such as stenosis of the pulmonalis, or patent foramen ovale or septum interventriculare, are designated cyanosis retinæ. The first case of the kind was reported by von Ammon,[2] observed in preophthalmoscopic days, in a twelve-year-old girl the subject of general cyanosis. Leber's first case[3] revealed no important heart-lesion post mortem, though enormous dilatation of the whole vascular system was found, while his second case exhibited congenital pulmonary stenosis.[4] The case which Liebreich de-

[1] Inaugural Dissertation, Dorpat, 1888.
[2] Klinische Darstellungen, 1841, Bd. iii. S. 75, Tafel XV.
[3] Verhandlungen des natur-historisch-medizinischen Vereins zu Heidelberg.
[4] Graefe und Saemisch, Handbuch der gesammten Augenheilkunde, S. 525.

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scribed and depicted[1] was due to stenosis of the pulmonalis, while Nagel's patient probably suffered from insufficiency of the pulmonary artery. Litten[2] speaks of a phthisical patient with cyanosis retinæ who, despite a complicated lesion of the pulmonalis and a defect in the septum interventriculare (a condition usually fatal in infancy), lived to the age of twenty-five years. Three other instances of cyanosis retinæ have been reported, two by Hirschberg and one by Knapp.

Ophthalmoscopically the veins may appear distended, tortuous, and of a purplish or violet-brown color. The arteries are rather serpentine, and of the color of normal veins. The sclera may be bluish or violaceous, or even pigmented, while the skin and mucous membranes are bluish gray and cold. The fingers are often misshapen. Vision is generally but little disturbed.

XVI. SPONTANEOUS PULSATION AND MANIFEST BLOOD CIRCULATION OF THE RETINAL VESSELS (PULSATIO SPONTANEA ET CIRCULATIO SANGUINIS MANIFESTA VASORUM RETINALIUM).

Spontaneous pulsation of the retinal arteries is never a normal condition. It may be seen in the retina when the normal blood-pressure falls below the normal intra-ocular tension, as in anæmia, chlorosis, syncope, the algid state of cholera, after profuse hemorrhages, and in senile arterio-sclerosis. It also follows compression of the central artery of the retina dependent upon diseases and tumors of the optic nerve and its sheaths or due to orbital tumors, and when the intra-ocular tension is increased while the blood-pressure remains normal, as in glaucomatous processes.

Another form of arterial pulsation consists in the continuation of the blood-waves out into the retina, sometimes to a considerable distance. This is observed in certain forms of chlorosis and anæmia, and in cases of insufficiency and aneurism of the aorta,—a fact of some clinical importance.

The first-mentioned variety, sometimes called pressure-pulse, is not necessarily an accentuated form of common arterio-retinal pulsation, although there may be variations in vessel calibre; but, as stated by Becker[3] and Raehlmann,[4] it is a phenomenon *sui generis*, which should be styled an "intermittent influx of blood" rather than a pulse. It is due to falling of the blood-pressure during systole below the intra-ocular tension, whether this be normal or reduced, while during diastole the blood-pressure can overcome the intra-ocular tension. We thus see retinal vessels filled during diastole and emptied during systole, constituting a true intermittent influx of blood through the arterial portal of the nerve head.

True arterial pulsation in the retina refers to abnormal extension of a high pulse-wave with rhythmical movements and calibre-changes, although complete interruption of the blood-stream never occurs. These calibre-

[1] Atlas, 1863, Plate IX. Fig. 3.
[2] Deutsche medicinische Wochenschrift, 1887, S. 144.
[3] Archiv für Augenheilkunde, 1872, Bd. xviii. S. 206.
[4] Klinische Monatsblätter für Augenheilkunde, Bd. xxviii. S. 1.

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variations are never associated with pressure-pulse. They are due not only to the diastolic dilatation of the vessels, but also to the *vis a tergo* of the blood-wave,—being therefore most easily seen in tortuous blood-vessels.

Although Helmholtz[1] sought for circulatory phenomena in the fundus oculi as soon as he had devised the ophthalmoscope, E. Jaeger[2] in 1854 was the first to observe pulsation of the retinal arteries. In the same year Donders[3] saw the same phenomenon, and von Graefe[4] noted it in glaucomatous affections. Later Wordsworth[5] observed the anomaly during syncope, and Graefe[6] recorded its occurrence with orbital tumors and descending neuritis. Since then numerous exhaustive contributions to the subject have appeared, based on clinical, anatomical, and experimental studies.

We can distinguish between two kinds of venous pulse in the eye,—physiological venous pulsation, the analogue of "arterial pressure-pulse," and progressive peripheral pulsation of the retinal veins, or true venous pulse.

Physiological venous pulse, though not seen in every eye, is not abnormal and does not ordinarily extend beyond the disk-margins. It has been explained as follows. Before the blood-wave in the retinal arteries passes through the capillaries to the veins, its influence is exerted upon the vitreous body, and the intra-ocular pressure is thus momentarily increased, so that the veins are most compressed at their exit, where the blood-pressure is lowest. Therefore the phenomenon is not an intrinsic venous pulse, but a rhythmic dilatation and contraction of the central vein dependent on the arterial pulse, though the latter is not visible. To quote Becker,[7] "The central vein is an index for the arterial pulse, and may be considered as a natural sphygmograph." This variety of venous pulse is often seen in diseases inducing increased intra-ocular tension, or it may be elicited in the normal eye by pressing gently on the eye with the finger.

Physiological venous pulsation was referred to as early as 1853 by Van Trigt[8] and Coccius,[9] and was first accurately described by E. Jaeger in 1854.[10] The explanation offered by Donders[11] in the same year is still the accepted one.

Progressive peripheral pulsation of the retinal veins is a true venous pulse, due to the passage of the blood-wave from the arteries to the veins, or to an increase of variations of pressure within the cavernous sinus. It

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[1] Beschreibung eines Augenspiegel.
[2] Wiener medicinische Wochenschrift, Nr. 2, 1854.
[3] Archiv für Augenheilkunde, Bd. i.
[4] Archiv für Ophthalmologie, Bd. i. S. 375.
[5] Royal London Ophthalmic Hospital Reports, vol. iv.
[6] Archiv für Ophthalmologie, Bd. x. S. 201.
[7] Loco citato.
[8] Nederlandisch Lancet, S. 465.
[9] Augenspiegel, S. 3.
[10] Wiener medicinische Wochenschrift, Nrs. 3, 4, 5.
[11] Archiv für Ophthalmologie, 1854, S. 75.

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is always pathological. It has been found associated with heart-lesions, especially aortic insufficiency, with arterio-sclerosis, and with certain forms of anæmia and chlorosis.

The rare phenomenon of manifest circulation in the retinal blood-vessels was first reported by E. Jaeger,[1] as seen in a man seventy-two years old who became blind over-night. The ophthalmoscope showed frequent interruptions in the blood-stream, which was of a dark color. During the momentary stases which occurred between the blood-waves the vessels were scarcely visible, while they could be easily recognized during each pulsation. Jaeger studied the condition for two weeks, when a beginning lenticular opacity shut off the fundus from view. It is probable that embolism of the central artery was the causative factor in this case. Liebreich[2] refers to a similar case seen in a detached retina, and Graefe[3] explains a similar venous disturbance, seen in cases of cholera and neuritis, as due to a lessened *vis a tergo*. The phenomenon is not infrequently found with embolism of the central retinal artery.

XVII. EMBOLISM OF THE CENTRAL ARTERY OF THE RETINA (EMBOLIA ARTERIÆ CENTRALIS RETINÆ).

By embolism of the central retinal artery is meant the complete or incomplete obstruction of this artery, or of one of its branches, from an embolus, which usually originates in a pathological process within the heart, or from an aneurism. The condition generally gives rise to sudden blindness of the affected eye and produces a characteristic ophthalmoscopic picture.

The first recorded case was observed by Jaeger,[4] but was not correctly understood by him.

The ophthalmoscopic diagnosis was made for the first time in 1859 by von Graefe in a man who became suddenly blind. It was doubted by many, but one and a half years afterwards the condition was proved by an autopsy. In 1861 similar cases were reported by Blessig[5] and by Schneller.[6] In the same year Liebreich[7] reported six cases. Since that time numerous uncertain cases have been seen. The last and most important example is one by Marple in which the embolus was found *in situ*.

I have seen thirty-six cases, fifteen in my clinic between 1883 and 1893 among sixty thousand patients. Of these, seven were treated in the clinic and eight came to the dispensary. Between 1867 and 1893, as Royal Provincial Oculist, I saw twenty-one cases among one hundred and twenty thousand patients, all in dispensary service.

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[1] Ueber Staar und Staar-Operation, S. 107.
[2] Archiv für Augenheilkunde, Bd. v. S. 261.
[3] Ibidem, Bd. xii. Ss. 145, 210.
[4] Loco citato.
[5] Archiv für Ophthalmologie, Bd. viii., 1, S. 216.
[6] Ibidem, S. 216.
[7] Berliner medicinische Gesellschaft.

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I. TOTAL EMBOLISM OF THE CENTRAL RETINAL ARTERY.

In this affection we may differentiate three distinct ophthalmoscopic pictures, according to the time which has elapsed since the embolism took place.

(*a*) *The Ophthalmoscopic Picture of Retinal Ischæmia.*—At once after the embolism occurs the fundus shows the highest retinal anæmia. The optic nerve head is pale white or yellowish white in tint, but never so shiny as it is in pure atrophy of the papilla. The arteries are filiform, and cannot be followed very far. In the large arteries, alone, sometimes extremely fine thread-like columns of blood may be seen. In other cases coagulated blood will be found in some of the peripheral branches, but cannot be traced to the main branches. The blood-vessel walls often have a white border. The veins are contracted, though to a much less extent than the arteries. They are narrower upon the optic disk and become relatively broader as they pass towards the ora serrata.

In the great majority of cases no pulsation can be found in the arteries or veins, even by marked pressure with the finger.

(*b*) *The Ophthalmoscopic Picture of Degeneration of the Retina.*—The optic disk is pale yellowish white. At times it is partially veiled by striped opacities. At the posterior pole of the eye the retina appears either white, milk-white, yellowish white, or greenish gray, and is more or less intensely opaque. This opacity is most pronounced in the macular region and along the larger blood-vessel trunks as they leave the nerve head. In a position corresponding with the macula a blood-red spot about one-third as large as the papilla can be seen. Usually it is of a regular transverse elliptical shape with sharp outlines, this being almost pathognomonic. This appearance, which diagnostically is so important, has in the course of years been explained in the most diverse ways. The oldest theory was that it was due to contrast between the whitish opaqueness of the retina surrounding the macula and the red fundus which could be seen through the macula, where the retina is the thinnest. This explanation was made by von Graefe[1] in the first case which he observed. The second theory explains it as a hemorrhage, either in the retina itself, as Blessig has assumed,[2] basing his conclusion particularly on the fact that the spot became darker and larger as the opaqueness of the retina increased, or situated behind the macula in the chorioid, and originating from collateral circulation in the territory of the short posterior ciliary arteries, as stated by Steffan.[3] A third theory, which probably has the least claim to probability, comes from Nettleship,[4] and explains this spot as a circumscribed central chorio-retinitis. Fischer[5] explains the red spot as due to the pigment of the

[1] Loco citato.
[2] Archiv für Ophthalmologie, Bd. viii., 1, S. 225.
[3] Ibidem, Bd. xii., 1, S. 46.
[4] Royal London Ophthalmic Hospital Reports, vol. viii.
[5] Ueber Embolismus, 1891.

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retina itself. According to Elschnig's opinion, which is based upon anatomical examination,[1] it is probable that both the chorioid and the retina take a part in its production,—the retina on account of the atrophy of its nervous elements and consequent attenuation, without any opacity such as is seen in the other parts of the retina (thanks to an increased nutrition from the chorio-capillaris, the net-work of which in this situation is developed to the highest degree), as well as a simultaneous atrophy of the pigment epithelium dependent upon the same anatomical peculiarity of this part. This, however, does not exclude the possibility that the intensity of the appearance may be enhanced by contrast and by the pigmentation of the macula itself.

In rare cases the cherry-red spot in the macula may be wanting, giving the same opacity at the posterior pole of the eye as in the remaining portions of the retina. Along the larger blood-vessels this opacity is generally more intense than in the other parts of the eye-ground.

The circulation of blood returns in the area of the central retinal arteries after embolism, usually happening in an irregular manner, as has been observed by Jaeger,[2] who described it as a visible circulation in the retinal blood-vessels. Sometimes the motion is reversed, contrary to the normal.

This interesting phenomenon of circulation is by no means seen so often as seems to be believed. It was recognized in the case of Jaeger,[3] then in the classic one of von Graefe,[4] then in Mayerhofer's case,[5] in von Hippel's,[6] and in a case seen by Hirschberg.[7] In most instances, however, although the circulation returns, this phenomenon does not appear. The return of circulation is probably best explained by the conclusions which Schnabel[8] has drawn from both clinical observations and anatomical researches. He found that the high degree of ischæmia of the retina immediately after embolism had occurred was not produced solely by the obstruction of the lumen of the blood-vessel by the embolus, but that as a second factor a spastic contraction of the blood-vessel walls was added. When, later, this spasm of the blood-vessel walls disappeared, the lumen of the artery was no longer totally obstructed by the embolus, and the blood might, even if only in a small quantity, again enter between the embolus and the vessel wall, and thus circulation be re-established.

When the circulation has returned, more or less numerous retinal hemorrhages may be observed. Formerly it was generally accepted that hemorrhages did not occur in total embolism, or at least occurred rarely and in small numbers, but that they appeared often and in large numbers in embolism of a branch. Schnabel[9] has, however, produced proof that in the

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[1] Archiv für Augenheilkunde, Bd. xxiv. S. 140.
[2] Loco citato.
[3] Loco citato.
[4] Loco citato.
[5] Inaugural Dissertation.
[6] Bericht.
[7] Centralblatt für praktische Augenheilkunde, 1885.
[8] Archiv für Augenheilkunde, Bd. xv. S. 28.
[9] Loco citato.

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cases examined by him hemorrhages occurred in an equal number of each kind. It was supposed that these hemorrhages were produced by a reversed blood-stream in the blood-vessels, the walls of which had been pathologically altered by the preceding ischæmia. It appears, however, that when the circulation is re-established the increased blood-pressure may be sufficient, even if it does not reach the normal, to rupture the badly nourished blood-vessel walls and to produce hemorrhages. Most of these hemorrhages are found in the macular region.

(*c*) *The Ophthalmoscopic Picture of Retinal Atrophy after Embolism of the Central Artery of the Retina.*—In this last stage the optic disk appears pale, dirty white, or yellowish white, giving an appearance to the nerve head similar to that seen in genuine atrophy. The nerve head, however, is generally more opaque, and approaches more nearly the type found in other forms of retinitis, as, for instance, so-called pigmentary retinitis and nephritic retinitis. It is sharply defined. The arteries are contracted to the highest degree, and become invisible as they pass towards the periphery. The veins are extremely narrow, and pursue a straight course.

Embolism of the central artery of the retina is often preceded by transitory obscurations of the visual field or by transitory blindness. These attacks may include but a part of the visual field, and may even appear in the field of vision of the fellow-eye. The embolic attack follows these prodromal signs either shortly or after weeks or months. Mauthner[1] has been fortunate enough to examine a case ophthalmoscopically during such an attack of transitory blindness, and he found the typical picture of ischæmia. The attack lasted an hour and a half, when suddenly the normal ophthalmoscopic picture and normal vision were re-established. From this occurrence Mauthner drew the correct conclusion that in this case the point of origin of the central artery of the retina from the ophthalmic artery had been blocked by a larger embolus, which on account of its size could not enter the smaller blood-vessel, and was subsequently carried farther by the blood-stream in the ophthalmic artery, to penetrate finally into some blood-vessel in which, on account of anastomoses, it produced no further symptoms.

The real total embolism always begins with an almost instantaneous, complete, and rarely curable blindness of the affected eye. The visual field is quickly obscured, and usually in a centripetal direction, from the periphery towards the optical centre. More rarely this takes place in a centrifugal manner. Often, however, it appears in a moment, so that the patient cannot give any information about it. Sometimes a small portion of the visual field, generally laterally situated, remains intact, but is also lost later, on account of the atrophy of the retinal elements. The same is the case, as a rule, with the improvements in vision which now and then result from a return of the circulation. Only when the ischæmia has not lasted long and the general affection takes a favorable turn can we hope for a lasting

[1] Loco citato.

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improvement. In the beginning light-flashes may be seen, but later they disappear when the atrophy becomes complete.

II. EMBOLISM OF ONE OR MORE BRANCHES OF THE CENTRAL RETINAL ARTERY.

If an embolus enters a branch of the central retinal artery, the same ophthalmoscopic picture results as that described in total embolism, except that the disturbance is confined to the area of the affected artery. If this happens in an inferior or a superior papillary artery, the characteristic appearance in the corresponding lower or upper half of the fundus will be found. If it occurs in a branch of these arteries only, changes in a sector of the fundus which corresponds to the area of that arterial branch will be seen. Ordinarily the embolized portion of the affected artery appears ophthalmoscopically as a spindle-shaped swelling, behind which the artery is filiform and almost empty. Subjectively, often the visual field seems obscured in the first moment of the embolism, as in total embolism. Gradually, however, it clears, and the visual disturbance remains confined to that part of the field which corresponds to the area of the affected artery. If this occurs in one of the papillary arteries, a superior or an inferior hemianopsia results. If it takes place in a smaller branch, a sector-shaped defect in the visual field will ensue.

Embolism of the macular arterioles is a well-characterized form of partial embolism. Perls[1] describes several such cases seen in Hirschberg's clinic which are interesting. It is well known that the macular arterioles are usually double, the superior and the inferior macular, which, as a rule, taking their origin from the central retinal artery in the papilla, become visible only on the temporal margin of the papilla, to pursue their course towards the macula. If, as happens not infrequently, one of these macular arteries becomes embolized, characteristic changes in the fundus confined to the macular region, or to a part of it, will be found, while the rest of the fundus will appear normal. This condition will produce a more or less extensive central scotoma with normal peripheral visual acuity.

III. EMBOLISM OF THE CENTRAL RETINAL ARTERY, WITH THE EXCEPTION OF THE MACULAR ARTERIOLES.

If an embolus has been driven farther into the central artery than where the two macular arteries originate, and these in consequence remain patent, the symptomatic conditions become reversed. The fundus is altered in the manner that is characteristic of an embolism, the macula alone remaining unchanged. The periphery of the visual field is darkened, while central visual acuity is preserved. Such an eye sees like one with considerable concentric contraction of the visual field, such as is found, for example, in pigmentary retinitis. Perls has described a case of this kind.[2]

[1] Centralblatt für praktische Augenheilkunde, 1890, S. 233.

[2] Ibidem, 1892, S. 161.

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IV. EMBOLISM OF THE CENTRAL RETINAL ARTERY COMPLICATED BY THE PRESENCE OF CILIO-RETINAL BLOOD-VESSELS.

When there are cilio-retinal blood-vessels, the entire fundus, with the exception of the area supplied by these cilio-retinal vessels, is changed in appearance to that which is so characteristic of embolism of the central retinal artery. Similar relative changes hold good concerning the disturbance of function. The visual field is reduced to the portion corresponding to the area of the functioning blood-vessels. When such cilio-retinal blood-vessels supply the macula (which is said to happen often, unless in such cases we have rather to deal with an abnormally deep origin of the macular arterioles), the picture presented is similar to the one described in the preceding paragraph.

Cases of this kind have been described by Benson,[1] Birnbacher,[2] and Hirschberg.[3]

In almost every case a uniform atrophy of the nervous elements is found in the retina,—that is, in its so-called cerebral layer. This is due to necrosis from want of arterial blood-supply. The nervous elements undergo a granular fatty degeneration. Later this detritus is absorbed, and simple atrophy results. The supporting fibres remain undisturbed. Elschnig denies the œdema of the retina which Hirschberg claims to have found. There are no true inflammatory symptoms and no emigration of cells. A proliferating arteritis of hæmatogenous origin, due to the ischæmia, has been seen. Elschnig found pathological changes of the pigment epithelium,—namely, atrophy and exudation of an albuminoid fluid, particularly in the macular region.

There is little or no difficulty in making the diagnosis of embolism of the central artery of the retina. It happens mostly in people suffering from some constitutional disease, the majority having organic change of the heart with hypertrophy or valvular lesion. Endocarditis, arterio-sclerosis, rarely Bright's disease, syphilis, and pregnancy, may all act as etiological factors.

Sudden blindness, with a characteristic ophthalmoscopic picture, renders the diagnosis certain. When no heart-disease or affection of the blood-vessels can be demonstrated, or if blindness occurs simultaneously in both eyes, care must be exercised in making a diagnosis.

Kern goes too far in maintaining[4] that in the majority of cases which present the picture of embolism of the central retinal artery no embolism has taken place, but that the ocular symptoms must have been caused by some local affections of the central artery, such as arterio-sclerosis or end-arteritis, due to syphilis, Bright's disease, or other dyscrasia; neither has he brought forward any anatomical proof.

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[1] Royal London Ophthalmic Hospital Reports, vol. x. p. 336.
[2] Centralblatt für praktische Augenheilkunde, 1883, S. 209.
[3] Loco citato.
[4] Inaugural Dissertation, Zürich, 1892.

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The prognosis of total embolism must, as a rule, be considered unfavorable. The most favorable course is observed in cases in which we succeed at a very early period in breaking up the embolus and in changing a total into a partial embolism. Everything depends on the patient's coming under treatment soon after the embolism has occurred, since the retina cannot withstand a prolonged ischæmia without being materially injured. Even in the proportionately favorable cases we usually see new embolisms follow, since the primary disease is not removed. The prognosis of partial embolism is somewhat better.

In recent cases the treatment consists in immediate and energetic massage of the eye, which should have been previously cocainized. This massage must be used for one or two minutes three times a day and continued for two weeks. The operative interference practised in former days, with a view of reducing intra-ocular tension, such as paracentesis of the anterior chamber, iridectomy, or sclerotomy, is no longer in use. Treatment must also be directed against the primary affection.

XVIII. SPONTANEOUS THROMBOSIS OF THE CENTRAL VEINS OF THE RETINA (THROMBOSIS VENARUM RETINALIUM SPONTANEA).

Spontaneous thrombosis of the retinal veins has thus far received but little attention. Leber[1] was the first to mention such an affection. Michel[2] published an extensive paper based on cases coming under his own observation. Then came two papers by Angelucci.[3] Later an inaugural dissertation by Wolff[4] made its appearance.

According to Michel, spontaneous thrombosis of the central retinal veins is usually observed in subjects between sixty and seventy years of age. It is mostly monocular, and most of the cases which have been observed have suffered from arterio-sclerosis. Some, in addition, had emphysema and cardiac hypertrophy. Like embolism, the affection appears suddenly, yet it is never preceded by any prodromal symptoms; neither does it reduce the visual acuity so much, or produce blindness, as does embolism. Vision may eventually improve materially, thus showing that the visual disturbances are slight as compared with the striking pathological changes seen in the fundus oculi.

Ophthalmoscopically, Michel distinguishes three degrees of the affection: 1, total thrombosis of the central retinal veins; 2, partial thrombosis; and, 3, simple stasis due to a very small thrombus.

In the first degree of intensity the optic nerve head and the surrounding retina appear suffused with blood. The outlines of the optic disk are blurred. Around this suffused portion there is a zone in which innumer-

[1] Graefe und Saemisch, Handbuch der gesammten Augenheilkunde, Bd. v. S. 231.
[2] Archiv für Augenheilkunde, Bd. xxiv., 2, S. 37.
[3] Klinische Monatsblätter für Augenheilkunde, 1878, S. 443, and 1879, S. 21.
[4] Würzburg, 1888.

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able circumscribed hemorrhages, varying in size, shape, and darkness of color, are found. In this part neither arteries nor veins can be seen. Where they become visible in the more peripheral portions, the veins appear very thick and tortuous and the currents assume a dark black-red tint. Sometimes a venous blood-column is seen to be interrupted, the part appearing yellowish white in color. The macula is whitish yellowish gray, the red spot in its centre being a real hemorrhage.

In the second degree of intensity the papilla is covered by radiary stripes of hemorrhage which tend towards the macula. This part of the retina corresponds with the suffused portion seen in the former degree. Externally from this zone similar numerous hemorrhages like those described above are found. The veins also appear changed in a similar manner, though not so markedly.

In the third degree but few hemorrhages are found. The arteries are thin. The veins, on the contrary, are very thick and tortuous and the currents are of a dark red tint.

In the course of the affection an organization of the thrombus may take place and lead to a total and lasting obstruction of the lumen; or a part of the lumen may become freed by a partial breaking up of the thrombus; or the thrombus may break up altogether and be removed.

In the first type, although in the beginning the symptoms seem to improve, later continually relapsing hemorrhages which penetrate the vitreous body, producing a total destruction of vision, take place.

In the other two types relapses may happen, each manifesting itself by a reduction of the visual acuity and the appearance of new hemorrhages. Finally, however, the fundus oculi improves and becomes clearer, whilst vision usually rises, for instance, from one-twentieth to one-fifth of normal.

During the course of the affection intra-ocular tension is never increased.

Michel groups these thromboses of the central veins among the marantic ones, basing this division upon a study of the macroscopical and microscopical conditions.

With regard to treatment, besides regulation of the diet and hygienic management of the eyes, Michel advises the use of Marienbad water, with twice weekly subcutaneous injections of one-milligramme doses of strychnine. Angelucci's[1] statements differ greatly from Michel's. He asserts that in his patients the blindness was at once complete, and that the subjects were all young people. He never saw retinal hemorrhages in his cases, and therefore presumes that they are not a constant symptom of marantic thrombosis. Personally, in spite of my enormous material for observation, I have seen but three cases,—one in my clinic among sixty thousand patients from 1883 to 1893, and two in my capacity of Royal Provincial Oculist among one hundred and twenty thousand patients from 1867 to 1893. They were all dispensary patients, and could be observed but cur-

[1] Loco citato.

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sorily. All of them were old subjects, and had arterio-sclerosis. What I saw fully agreed with Michel's description.

XIX. RETINAL HEMORRHAGES, RETINAL APOPLEXY (HÆMORRHAGIÆ RETINÆ, APOPLEXIA RETINÆ).

Hemorrhages appear in the retina either as collateral symptoms in diverse forms of retinitis or spontaneously during different constitutional diseases or affections of the eyeball. They also follow various traumatisms. They are generally due to alterations in the blood-vessel walls or in the blood itself.

In the ophthalmoscopic picture they appear as light or dark red spots, which may have a whitish rim, or may be transformed into whitish foci of degeneration. In very rare instances they may be changed into pigment. Their size, shape, number, and situation vary greatly. Their size ranges from a barely visible point to an extravasation which occupies the entire retina. Their shape and situation are also extremely variable. When they lie in the nerve-fibre layer they are striped or flame-like and have a radiary arrangement. When they lie in the deeper layers or are situated at the outer or the inner surface of the retina they appear more or less roundish, oval, or irregular in shape. They are distributed mostly along the course of the blood-vessels. They may extend over the whole of the retina or occupy its more peripheral portions, or may be localized at the posterior pole. In the macular region large characteristic extravasations can often be observed.

The amount of visual disturbance depends on the locality, size, and number of the hemorrhages, a defect in the visual field corresponding to each. If they lie in the macula the central visual acuity is generally abolished; when they are small and peripherally situated they may disturb vision very little or not at all. Sometimes metamorphopsia and very rarely photopsiæ are complained of.

Retinal hemorrhages are frequently observed, and are seen under greatly varying conditions. Spontaneous retinal hemorrhages may occur in patients with perfect or relatively normal general health. An increase of blood-pressure in general plethora, excessive physical labor, hard coughing-spells and vomiting, and especially childbirth, may produce them. Bathing in too hot water is also mentioned as a cause. Hypertrophy of the heart will aid in their production. They may occur when the efflux of the blood is interfered with by an increase of the intra-ocular pressure upon the venous exit, as in glaucomatous conditions; to this probably should be added a degeneration of the blood-vessel walls. They appear when there is a compression of the central retinal vein in the optic nerve by diverse pathological processes, particularly tumors, and also, though more rarely, when there are obstacles to the circulation in the general venous apparatus.

In new-born children retinal hemorrhages have been frequently found.

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Königstein[1] first directed attention to this fact. In two hundred and eighty-one cases he saw twenty-nine with retinal hemorrhages,—that is, about ten per cent. Schleich[2] found among one hundred and fifty children examined by him for the same purpose (the youngest of whom were just born, while the oldest was eight days old) retinal hemorrhages seventy-eight times in forty-nine cases,—that is, in about thirty-three per cent. Bjerrum[3] determined their existence in three per cent. only. Naumhoff[4] examined the eyes of forty-seven corpses of new-born children, and found pathological changes in twelve cases,—that is, in about twenty-five and a half per cent. These hemorrhages are mostly found at the posterior pole of the eye, are monocular or binocular in type, sometimes roundish or irregular, and sometimes striped and radiary. They are quickly absorbed without leaving a trace. It may be that by such hemorrhages many an amblyopia without ophthalmoscopic symptoms can be explained. Their cause is probably the increase of the intra-cranial pressure during birth by compression of the skull.

Anomalies of menstruation, amenorrhœa, and the climacteric changes produce retinal hemorrhages, though rather seldom. The first of such cases was described by Liebreich[5] in 1854, and was seen in von Graefe's clinic. It appeared in a woman during the climacteric period. With the ophthalmoscope a large rhomboidal hemorrhage could be seen in the region of the macula. A second and somewhat similar case was reported by the same author[6] in 1863.

In 1862, so far as I can remember, I saw, while I was assistant to the late Professor Hasner, an extremely interesting type which differed entirely from the others that have been observed.

The case was that of a sixteen-year-old Jewish girl. She was sent to the clinic on account of conjunctival hemorrhages. She had had amenorrhœa for several months. I thought the enigmatical conjunctival hemorrhages indicated a case of malingering, especially since she came from an independent medical clinic, in which at that time particular attention was being paid to all sorts of hysterical affections and seeming diseases, among which simulated ones were not lacking.

I was called a number of times to see these hemorrhages, but, although answering the calls at once, I never found anything else than the bloody traces of an alleged preceding hemorrhage; and thus my suspicion of simulation was considerably strengthened.

In order to settle the question, I stationed two nurses at the patient's bedside, and ordered that they stay there day and night and watch the

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[1] Wiener medicinische Jahresbericht, 1881, S. 47.
[2] Mittheilungen aus der Klinik, Tübingen, 1884, Bd. i. S. 44.
[3] Congress, Copenhagen, 1884.
[4] Archiv für Augenheilkunde, Bd. xxxvi., 3, S. 180.
[5] Archiv für Ophthalmologie, Bd. i., 2, S. 206.
[6] Loco citato.

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patient and control every movement. On the second day I was called to see a hemorrhage which was just starting, and then I witnessed how the blood oozed from the conjunctiva of both eyes, especially from the retro-tarsal fold, as if a sponge soaked with blood was being slowly squeezed out. I repeatedly cleansed the conjunctival sac of blood, and at once a new layer of blood covered the surface, gradually filling the conjunctival sac. This interesting phenomenon lasted a minute and a half, and occurred again twice on the same day; the next day it occurred four times, the third day once, after which it occurred no more during the patient's stay at the clinic. I was convinced, therefore, that this was no simulation, but a spontaneous vicarious conjunctival hemorrhage at a period when the patient expected her menses.

The ophthalmoscope revealed in both eyes the astonishing conditions shown on Plate VII. In the periphery of the retina, near the ora serrata, I found innumerable small hemorrhages. The posterior pole and the region of the papilla and macula were free from them, which was contrary to every case thus far observed of retinal hemorrhages due to menstrual anomalies. Central visual acuity was intact, and peripheral vision was but slightly reduced. I ordered iron, with rest in bed. In about three weeks after her menses had appeared she left the clinic. I never saw her again. All such cases of retinal hemorrhage thus far observed have pursued a favorable course.

Sometimes retinal hemorrhages have been seen during pregnancy and the puerperal state. Such cases, in which total blindness usually resulted, have been reported by Critchett,[1] Litten,[2] and Doepner.[3]

In patients between sixteen and twenty-four years of age, of both sexes, such hemorrhages have been observed without any known cause. In these cases relapses frequently occur. The hemorrhages are often numerous, more or less extensive, situated peripherally or in the macular region, and are frequently complicated by hemorrhages into the vitreous body. They may eventually lead to proliferating retinitis or to secondary glaucoma.

Hemorrhages are also found in very old people who are suffering from arterio-sclerosis or from miliary aneurisms and are simultaneously disposed to cerebral apoplexy, as has been pointed out by Boucheron and Charcot.

Spontaneous retinal hemorrhages have been seen during severe constitutional diseases, such as scurvy, purpura hæmorrhagica, pyæmia, septicæmia, pernicious anæmia, and, more rarely, in syphilis and icterus.

Retinal hemorrhages as part symptoms during diseases of the retina and optic nerve may appear, as, for example, in embolism of the central retinal artery or its branches, in thrombosis of the retinal vessels, in hemorrhagic nephritis, in leukæmic, diabetic, and specific retinitis, and in optic neuritis.

Retinal hemorrhages may also be due to poisoning by phosphorus and

[1] Royal London Ophthalmic Hospital Reports, vol. vii. p. 38.
[2] Centralblatt für praktische Augenheilkunde, 1877.
[3] Ibidem.

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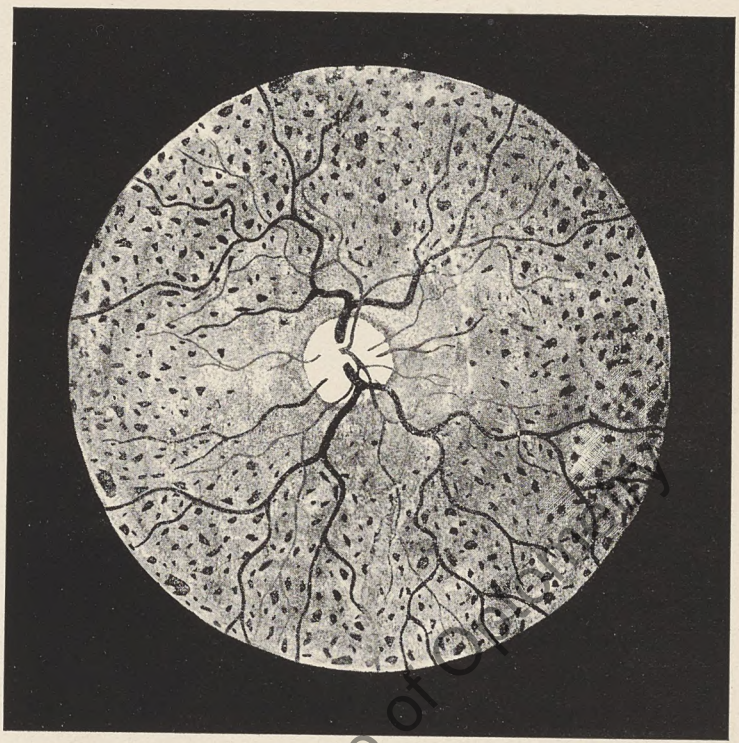
PLATE VII.



Ophthalmoscopic appearance of retinal hemorrhages due to menstrual disturbances.

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lead. Rarely they are found as complications to the toxic effects of arsenic, carbonic oxide gas, sewer-gas, etc.

Traumatic retinal hemorrhages, in which the continuity of the blood-vessel walls is interrupted, may arise from shooting, cutting, striking, and stabbing, ruptures of the sclerotic from injury with blunt instruments, contusions of the eyeball and isolated ruptures of the retina, severe contusions of the head, as by falls from great heights without direct injury to the eye, penetration of foreign bodies into the eye, and operations, such as iridectomy for glaucoma or secondary glaucoma.

They usually run a protracted course, sometimes several months elapsing before they are absorbed. Frequently perfect restoration is observed, especially when no relapses have taken place. In unfavorable cases a gradual contraction of the retinal blood-vessels, with consequent atrophy of the retina and the optic disk, may result. In rare instances a pathological formation takes place in the residua of such hemorrhages, or connective-tissue membranes which form the basis of retinitis proliferans may develop.

In the treatment the etiological factors must be dealt with before we can think of curing the affection itself. Perfect rest, both bodily and mental, is necessary. The patient must remain in bed in a darkened room. Locally, cool compresses should be ordered, followed by local depletion or the use of a Heurteloup artificial leech. At times atropine is useful. Cathartics are indicated. Mercurial inunctions and iodide of potassium can be employed with benefit. Diaphoresis by means of subcutaneous injections of pilocarpine is of service. Menstrual anomalies can be best cared for by such roborants as iron and quinine.

XX. HEMORRHAGIC RETINITIS AND HEMORRHAGIC NEURO-RETINITIS (RETINITIS HÆMORRHAGICA ET PAPILLO-RETINITIS HÆMORRHAGICA).

Although retinal hemorrhages occur in many forms of retinitis, yet only cases in which numerous ones are found in an inflamed retina without the presence of any intercurrent disease are recognized as hemorrhagic retinitis. On the other hand, such patients generally suffer from chronic affections of the organs of respiration or of the circulatory apparatus; especially from diseases of the heart and blood-vessels.

Even in preophthalmoscopic times we find some reports which undoubtedly refer to hemorrhagic retinitis. As Leber states, in 1846 von Seidel and Kauka[1] presented a communication concerning the combination of amaurotic conditions with diseases of the heart and larger blood-vessels. In 1851 Blodig[2] made an analogous statement.

In 1855 Liebreich[3] gave the first accurate description and a picture which was true to nature.

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[1] Medicinische Jahrbücher des kaiserlich-königlichen österreichischen Staates.
[2] Zeitschrift für die gesammte Wundärzte.
[3] Archiv für Augenheilkunde, Bd. i.

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In 1856 von Graefe[1] reported the condition of the visual field in such cases; and the same year E. Jaeger[2] gave an illustration of the affection. Five years later Mandelstamm[3] reported several cases. This was followed in three years' time by Galezowski's[4] assertion of the monolateral occurrence of this disease. In 1871 Power[5] saw a case of monolateral hemorrhagic retinitis with small hemorrhages surrounding the papilla and large peripheral extravasations. He also infrequently found small white spots in the retina with a star-like figure around the macula, as in albuminuric retinitis, though he expressly states that the urine contained no albumen. In 1874 Hutchinson[6] studied the affection and also mentions a monocular case. Three years later Leber[7] directed attention to the monolateral form of the disease and noted the sudden appearance of visual disturbances. From this he concluded that neither hypertrophy of the left ventricle nor rigidity of the walls of the large arteries could be the sole cause of the trouble, as had till then been supposed. He believed that multiple embolisms in the smaller branches of the central artery of the retina were the cause of the affection; and this opinion was later corroborated by an anatomical examination by Wagenmann.

In 1878 Michel[8] found anatomically thrombosis of the central retinal vein in a case of hemorrhagic retinitis. He recognized three degrees of intensity of this affection, and gave an ophthalmoscopic representation of each.

In the first, he says, there is occlusion of the central vein and the papilla, and the surrounding retina appears suffused with blood. The outlines of the papilla are invisible. Outside of the suffused zone there are numerous small hemorrhages varying in size and shape, some being light red and others dark red in tint. The arteries are thin; the veins are enormously dilated and very tortuous, and the currents are dark black-red in color. The blood-column in the veins appears sometimes interrupted. The macula shows a yellowish-gray discoloration, and in its centre there is a hemorrhage. In the case studied, blindness came on suddenly and light-perception alone was retained.

In the second degree of intensity, in which the lumen of the central vein is only partially occluded by the thrombus, the outlines of the papilla are hidden by hemorrhages in stripes which extend in all directions. The arteries are barely visible. The veins, however, appear as they did in the former condition. Outside of the peripapillary zone of hemorrhages in stripes there are a great many which differ in both size and shape. In

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[1] Archiv für Augenheilkunde, Bd. ii.
[2] Beiträge zur pathologische Augenheilkunde.
[3] Pagenstecher's Klinische Mittheilungen.
[4] L'Union médicale.
[5] Royal London Ophthalmic Hospital Reports.
[6] Ibidem.
[7] Graefe und Saemisch, Handbuch der gesammten Augenheilkunde.
[8] Archiv für Augenheilkunde, Bd. xxiv.

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this degree vision is less interfered with, yet later it gradually grows less, and at the same time new and recurrent hemorrhages can be observed.

In the third stage, when the thrombus occludes but little of the lumen of the central vein, there are few hemorrhages present. Here, however, there is a great disproportion between the diameter of the arteries and that of the veins. The arteries are thin, while the veins are very much dilated and tortuous. The hemorrhages in the papilla and its neighborhood are in stripes; near the periphery they are larger and of different shapes. Vision is less disturbed, and may return to the normal. There are small point-like opacities in the vitreous. Increase of intra-ocular tension has not been observed.

Wagenmann[1] reports three interesting cases of hemorrhagic retinitis in which anatomical examinations were made. The first case was in a patient who suddenly became blind in one eye. The ophthalmoscope revealed only a red papilla with indistinct outlines. The retina appeared opaque for quite a distance, and was strewn with a multitude of small and medium-sized hemorrhages reaching as far forward as the ora serrata. Near the papilla these hemorrhages were arranged in radiary stripes; the remainder were round. In places the arteries, which were covered with hemorrhages, showed no material changes, nor could pressure produce pulsation. The veins were bordered by opaque lines which sometimes swelled to white plaques. They were dark brown-red, and were greatly dilated and very tortuous. In this case the sudden blindness and the conditions of the arteries indicated embolism of the central artery, while the multitude of hemorrhages seemed to show that the condition was thrombosis of the central vein. The anatomical examination showed a high degree of narrowing, and in some places a complete obliteration, of the retinal arteries by endarteritis.

It seems clear, therefore, that the sudden embolic occlusion did not occur in the main trunk of the central artery, but in the several branches within the retina, just as Leber[2] had supposed.

In the second case, in which the visual disturbance developed gradually and the ophthalmoscope revealed a red papilla with indistinct outlines and numerous flame-shaped hemorrhages but no white foci of degeneration, he found anatomically a thrombus which partially occluded the central vein some distance behind the eyeball.

This interesting case is the anatomical proof of the second degree of intensity of thrombosis, as described by Michel.[3]

In the third case the patient had been ailing for some time, and the eye was finally enucleated on account of a hemorrhagic glaucoma. Ophthalmoscopically it showed, besides hemorrhages and inflammatory signs in the retina, numerous yellow foci of degeneration.

[1] Archiv für Augenheilkunde, Bd. xxxviii.
[2] Loco citato.
[3] Loco citato.

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A one-sided inflammation of the vascular system of the retina was found, without any known cause, which, aside from the retinitis, had led to numerous hemorrhages due partly to changes in the blood-vessel walls and partly to multiple arterial and venous thromboses of the retinal vessels without thrombosis of the central vein.

The ophthalmoscopic findings vary greatly. The optic disk is swollen, opaque, has indistinct outlines, and is often covered with striped hemorrhages. In many cases the swelling is insignificant. In others it is moderate, and in some instances it may reach the highest degree of genuine papillitis, so that the nerve head protrudes into the vitreous over the surrounding retina, like a mushroom. In other words, all degrees of swelling and inflammation, from a simple retinitis to a hemorrhagic papillo-retinitis, may be present.

The redness of the optic disk is also variable. Often it is but moderately reddened. Again, its redness may be so considerable that it cannot be distinguished from the surrounding fundus. Sometimes the outlines of the papilla are only veiled; at other times they are invisible, so that the position of the disk can be judged only by the grouping of the larger retinal blood-vessels. The stripe-like hemorrhages which at times lie partly or altogether in the papilla likewise differ as regards their number and extent. The retina is somewhat swollen and opaque, especially around the papilla. Towards the periphery this dimness reaches to an irregular distance. The retinal hemorrhages are variable as regards their number, size, shape, and distribution. They appear ophthalmoscopically as light or dark red spots. Sometimes but one hemorrhage, or a few only, can be seen, but there are cases in which they are multitudinous. Their size varies between a barely visible point and an area which covers almost the whole retina. Their shape when situated in the nerve-fibre layer is the usual one,—*i.e.*, that of stripes. Sometimes they are flame-shaped and show, especially near the papilla, a radiating arrangement. The deeper-seated ones are mostly round, though they may assume all possible shapes. In typical cases striped or flame-shaped ones are found near the papilla, and sometimes upon it. Near the ora serrata larger round ones seem to be more prevalent. When their number is small and they are distributed over the whole area of the retina, points of predilection (usually the neighborhood of the papilla, the course of the larger blood-vessels, and the macular region) are found. In some instances they appear on the inner or the outer retinal surface, and rarely they break through into the vitreous.

Yellowish-white spots, due to fatty degeneration, appear probably near the hemorrhages only. In rare cases shining white areas, due to varicose hypertrophy of nerve-fibres, are seen, but they are always present in small numbers in comparison with the hemorrhages. In most cases neither of these kinds of spots can be observed. The foci of fatty degeneration are usually whitish yellow, and, since they generally lie in the posterior layers of the retina, blood-vessels can be seen passing over them. The plaques,

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due to degenerated nerve-fibres, are, of course, located in the nerve-fibre layer, are pure white, have at times a fatty sheen, and infrequently hide some of the smaller retinal blood-vessels.

The arteries are either normal or slightly narrowed. Sometimes single branches are filiform, or are changed to white bands and obliterated. The veins are greatly dilated and carry very dark-colored blood. They often appear interrupted, because in their tortuous course they at points dip farther into the tissue and are masked by the opaque retina. Sometimes the interruption is the result of an absence of circulatory movement, the stagnating and coagulated blood-column being seen by the side of an empty and apparently obliterated portion of the vessel. In some cases the veins (more rarely the arteries) are bordered by white lines. On account of the absence of circulation in certain blood-vessels, that part of them which lies on the papilla cannot be emptied by pressure, and therefore the phenomenon of pulsation cannot be produced.

The following complications have been observed: hemorrhages into the vitreous; vitreous opacities; hemorrhagic glaucoma; disseminate chorioiditis, iritis and irido-cyclitis, and amotio retinæ. Distant complications consist in hemorrhages in different organs, due to arterio-sclerosis, especially cerebral hemorrhage with its consequences.

Generally the visual disturbance appears suddenly in one eye, and is usually noticed in the morning on awakening.

Sometimes, in fulminant cases, total blindness may at once result, or vision may sink to mere light-perception. In other cases the visual disturbance appears gradually, often spasmodically, owing to reiterated occurrence of new hemorrhages. The amount of visual disturbance depends on the number, the size, and the localization of the hemorrhages. The gravest disturbances are due to macular hemorrhages. Even small hemorrhages in this place materially reduce central vision. When they are larger, central scotomata result. Peripheral ones, unless they are extensive, impair eccentric vision but slightly, consequent contraction of the visual field, metamorphopsia, photopsia, and chromopsia being but rarely seen.

Anatomically, we find as the most important causes of hemorrhagic retinitis spontaneous thrombosis of the central vein, multiple thrombosis, or embolisms of the smaller retinal blood-vessels. The alterations in the blood-vessel walls from endarteritis, as described by Michel and Wagenmann,[1] have been mentioned.

Aside from this we often find that the papilla and the retina are œdematous and infiltrated with round cells. The hemorrhages form a hemorrhagic infiltration of the retina, so that the blood-corpuscles lie between its different elements and cause it to seem swollen. In the nerve-fibre layer they extend along the nerve-fibre bundles, and therefore appear as stripes

[1] Loco citato.

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or flame-like bodies. In the deeper layers they conform more to the supporting radiary fibres, and therefore appear more round or polymorphous. Large hemorrhages sometimes destroy the retinal tissue and either project towards the vitreous or penetrate into it or into the outer surface of the retina, destroying the rods and the cones and producing a localized hemorrhagic amotio retinæ.

Hæmatogenous pigment is seldom seen. Connective-tissue scars are formed only in consequence of large hemorrhages where there has been much destruction of retinal tissue. The central artery and its branches often show the signs of a grave endarteritis, while the alterations in the veins are usually insignificant.

The course of the disease is slow. Although it may make its appearance in a fulminant manner and destroy sight in a moment, yet in most instances the symptoms manifest themselves gradually. Temporary improvements may be observed, only to be lost through the occurrence of new hemorrhages. Later the color of the optic disk generally assumes an atrophic tint and the arteries grow so attenuated that in places they are barely visible. In some instances an increase of intra-ocular tension—a true hemorrhagic glaucoma—may develop, this, as a rule, quickly destroying any remaining vision. Again, a cerebral apoplexy may produce death.

Almost without exception the condition attacks but one eye. It is usually found in subjects from the fortieth to the sixtieth year, and most frequently afflicts those who suffer from arterio-sclerosis with hypertrophy of the left ventricle. However, it is seen also in other affections of the heart and the larger blood-vessels, such as valvular lesions, aneurisms of the large arteries, etc.

In females it may be produced by sudden suppression of the menses, or may develop in the climacteric period. It is seen in hemorrhoidal subjects suffering from cessation of the hemorrhages; also in intoxication from phosphorus or nitrobenzol. In the latter case the entire fundus is of a violet tint and the blood-vessels appear as if filled with ink. It has also been observed after severe burns of the skin, and, according to von Knies, is seen after intermittent fever.

There can be very little doubt as to whether affections of the heart and of the blood-vessels favor the occurrence of hemorrhages in the retina giving rise to hemorrhagic retinitis; that, however, these alone do not suffice as etiological factors is proved by Leber's researches concerning this point.

Diagnosis is not difficult when it is possible to exclude the presence of general systemic diseases, such as sepsis, nephritis, diabetes, leukæmia, pernicious anæmia, syphilis, etc., and when diseases of the heart or blood-vessels or other etiological factors can be found. If this is not the case, it is often impossible to give an answer from the ophthalmoscopic picture alone, since in the diseases above enumerated similar or identical pictures

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in the fundus oculi may be found, and since even the relative proportion of the white plaques to the numerous hemorrhages is of no diagnostic importance. Hemorrhagic retinitis differs from a simple retinal hemorrhage by reason of the presence of true inflammatory symptoms, by the swelling and dimness of the papilla and retina, by the indistinctness of the outlines of the papilla, and by the alterations in the blood-vessels.

In general, prognosis is unfavorable. Cure is possible only when the macular region has remained free and none of the larger retinal arteries have become obliterated; this, however, is rare. As a rule, the absorption takes a long time, being complicated by recurrent hemorrhages, until vision is finally reduced.

If hemorrhagic glaucoma does not develop, the hemorrhages may slowly disappear, leaving behind spots of hæmatogenous pigment. The papilla then shows the signs of retinitic atrophy. The blood-vessels grow thin and become partly obliterated.

Treatment of hemorrhagic retinitis is usually symptomatic. All noxious influences by which the blood-pressure is increased and a congestion towards the head produced must be avoided. Abstinence from spirituous liquors and from other stimulants must be observed. Mental or bodily exertions or excitements are to be avoided. The heart's action should be regulated by digitalis. Local depletions, so often recommended, should be made with great caution, and then only in very plethoric individuals. The diet is to be regulated and defecation promoted. Iodide of potassium should be administered internally, and foot-baths should be ordered. In recent cases it is advisable to keep the patient in bed in a moderately dark room. The head should be kept high, and cold compresses applied to the forehead and temples. Later a careful diet must be insisted on. The eyes should have rest behind dark glasses. Ergotin internally and strychnine by injection into the temples are to be recommended. Inunctions of Arlt's ointment or of iodide of potassium ointment to the temples may be useful.

XXI. HYPERPLASTIC RETINITIS (RETINITIS HYPERPLASTICA).

Hyperplastic retinitis[1] I consider as a more or less inflammatory process in the retina, which, in the manner of hyperplastic formations in other connective-tissue organs, leads gradually either to a more or less prominent and tumor-like hyperplasia or to the formation of neoplastic bands or membranes in the whole or a part of the retina. This occurs when, in consequence of emigration, proliferation, and organization of round cells, with the formation of neoplastic blood-vessels (to which may be added organization of hemorrhages and proliferation of tissue), granulation material is produced. Three groups of this interesting and highly characteristic affection can be recognized.

[1] Archiv für Augenheilkunde, Bd. xx. Ss. 98, 114.

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I. HYPERPLASTIC HEMORRHAGIC RETINITIS, HEMORRHAGES INTO THE RETINA OR THE VITREOUS BODY (RETINITIS HYPERPLASTICA HÆMORRHAGICA, HÆMORRHAGIA RETINÆ AUT CORPORIS VITREI; BINDEGEWEBSNEUBILDUNG IM GLASKÖRPER, JAEGER; PRORETINALE BINDEGEWEBSNEUBILDUNG, HIRSCHBERG; RETINITIS PROLIFERANS, MANZ).

This disease, which is the result of hemorrhages into the retina or the vitreous body, with its characteristic ophthalmoscopic picture, was seen for the first time in 1864 by Mackenzie[1] in a patient who was suffering from oxaluria. Doubtless, however, there was no connection between the oxaluria and the retinal disease. In 1867 Becker[2] observed a remarkable case, in which the vitreous body was more affected than the retina, and in which products of hemorrhages were probably the starting-point for a vascularized connective-tissue formation the blood-vessels of which communicated with those of the retina. Later the eyeball degenerated. In 1869 Jaeger[3] observed a similar case, and depicted it under the name of new formation of connective tissue in the vitreous body. In 1874 Hirschberg[4] published an example of the affection, and called it proretinal new formation of connective tissue. In 1876 Schnabel[5] described such a case in a patient twenty years old, who, having had very weak eyes previously, suddenly became blind over-night, and in whom the next morning impenetrable opacities could be seen in the vitreous body (undoubtedly hemorrhages). From this condition the picture of a hyperplastic hemorrhagic retinitis developed. Soon after Manz published three such cases,[6] and termed them retinitis proliferans, which name has since been accepted. He also saw in his cases hemorrhages in the retina and the vitreous body, though he denies a connection between these and the characteristic retinal affection. He believes them to be immaterial and secondary. The eyeball of one of these cases was enucleated on account of great pain, and the author[7] reported the anatomical conditions. The eyeball was, however, undergoing progressive atrophy from irido-chorioiditic processes, and the examination proved therefore to be of slight value. In 1877 Leber[8] published several instances, and was the first to suggest that these retinal new formations developed from hemorrhages into the retina or the vitreous body. In 1884 Alexander[9] reported such a case in which hemorrhages still existed, and which un-

[1] The Ophthalmic Review.
[2] Berichte der Augenklinik zu Wien, S. 106.
[3] Beiträge zur Pathologie des Auges, Tafel lx.
[4] Klinische Beobachtungen.
[5] Archiv für Augenheilkunde, Bd. v. S. 124.
[6] Ibidem, Bd. xxii., 3, S. 229.
[7] Archiv für Ophthalmologie, Bd. xxvi , 2, S. 55.
[8] Graefe und Saemisch, Handbuch der gesammten Augenheilkunde, Bd. v. S. 665.
[9] Deutsche medicinische Wochenschrift, Nr. 40.

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doubtedly developed from them. In 1890 Schleich[1] published two instances which are of great importance, since they were kept under direct clinical observation from the beginning of the disease to the complete formation of connective tissue in the retina, and therefore proved that the formations developed from preceding hemorrhages. At the same time Proebsting[2] published a case which was complicated with albuminuric retinitis. In 1892 von Hippel[3] saw a case in which it was impossible to demonstrate previous hemorrhages, but in which there were retinal changes and neoplastic blood-vessels that penetrated into the new formation. In 1893 Blessig[4] made a communication upon a case in which hemorrhages could be seen in the fellow-eye, while in the eye with preretinal new-formed connective tissue their previous occurrence was rendered very probable. In the same year Axenfeld[5] reported a case which was complicated with rupture of the retina, and in which hemorrhages had previously occurred.

I have seen only two typical cases of this affection at my clinic from 1883 to 1893, among fifty-nine thousand and seventy-seven patients. One of them, a man aged forty-five years, appeared but once in the dispensary, so that a very accurate description of the typical and characteristic ophthalmoscopic changes cannot be given. Eight years previously he had been treated for secondary syphilis. The appearance of previous hemorrhages into the vitreous body could not be demonstrated. The second, a boy aged nine years, remained under observation for more than two months. This case is of interest, since hemorrhages into the vitreous body as well as preretinal new formation of connective tissue were found in both eyes, rendering it unique, so far as I know. Unfortunately, the clinical report of this case, as well as the drawings appertaining to it, was stolen from the clinic. In my practice as Royal Provincial Oculist I have, among one hundred and twenty thousand eye-patients, from 1867 to 1893, seen but three typical cases of this rare affection. One of these was in an official fifty years old, a heavy drinker; the second was in a seamstress forty years old; the third I saw in a miner thirty-six years old. Each came but once to the dispensary. In all, previous hemorrhages could be demonstrated with some degree of certainty.

No doubt can remain that in most, if not all, of the cases of this type there existed a connection between hemorrhages into the vitreous body and the retina and the new formation of connective tissue in the retina. In at least ninety per cent. of all reported instances, simultaneous or previous hemorrhages could be proved with more or less certainty, and in the few in which this was not the case the possibility of a previous hemorrhage could not with any certainty be excluded.

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[1] Klinische Monatsblätter für Augenheilkunde, Bd. xxviii. S. 63.
[2] Ibidem, S. 73.
[3] Ibidem, Bd. xxx. S. 370.
[4] Ibidem, Bd. xxxi. S. 202.
[5] Archiv für Augenheilkunde, Bd. xxvi. S. 225.

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Ophthalmoscopically, the membranes in the retina impressed every observer as new-formed connective tissue, as is shown by the names given to the condition, such as new formation of connective tissue in the vitreous body (Jaeger), proretinal new formation of connective tissue (Hirschberg), preretinal formation of connective tissue (Blessig), etc. In most cases neoplastic pathological blood-vessels were found in these connective-tissue membranes.

With the exception of Proebsting's and von Hippel's cases, none of the reported instances have shown any symptoms of retinitis, nor even of hyperæmia of the unaffected parts of the retina. On the contrary, the unaffected portions of the retina were normal, with a corresponding degree of visual acuity. Further, in Proebsting's case there was an accidental complication with nephritic retinitis, and in von Hippel's case the nephritic symptoms were immaterial, and probably but an accidental complication. Moreover, in all the anatomical examinations, aside from proliferation of the retinal tissue, a new formation of connective tissue, with sometimes one of neoplastic blood-vessels, was the main change. Yet none of these examinations could be considered as having been made on a pure case of recent hyperplastic hemorrhagic retinitis, since one was altered by a severe irido-cyclitis, another was found in a phthisical eyeball, and a third was of traumatic origin and thus did not belong to this group.

We must further bear in mind that it is hardly probable that a spontaneous inflammation (a so-called retinitis proliferans) would confine itself to so small and so well defined a portion of the retina, be of so queer a shape, and sometimes present sharply cut gaps in which the retina appears normal. Finally, we must not overlook the fact that similar membranous connective-tissue formations with neoplastic blood-vessels and of similar irregular shapes have been frequently observed and described as the results of hemorrhages into the vitreous body.

Similar formations may take place in hemorrhagic glaucoma, as Deutschmann has observed; and very extensive membranous formations with large retinal hemorrhages and hemorrhages into the vitreous body may occur after severe traumatisms, as described by Cohn and examined microscopically by Waldeyer.

From this it follows that a primary retinal affection must be excluded, and that we are not dealing with a retinitis proliferans. The condition is a secondary affection of particular portions of the retina, which are stimulated to proliferation in their inner layers by the irritation caused by the presence of the hemorrhages. To this must be added the organization of the hemorrhages, cell-proliferation, and formation of new blood-vessels, by which a true process of granulation, which leads to the formation of neoplastic connective tissue, is inaugurated, this finally changing into scar-tissue,—a true hyperplastic retinitis.

It is useless to ask why similar new formations of connective tissue are not observed after all hemorrhages into the retina or the vitreous body, since

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such questions might be asked in other ophthalmological and general conditions without the possibility of an answer.

The ophthalmoscopic picture of hyperplastic hemorrhagic retinitis is so characteristic that whoever has seen it but once can never forget it, nor confound it with that of any other affection.

At the posterior pole, covering a more or less extended part of the retina (sometimes also the optic disk, but rarely the peripheral parts), can be seen a shining white, a bluish-white, or a yellowish-white membrane. Sometimes it will be found with one or more sharply defined gaps, which differ in size and shape, being round or oval or crescentic, and through which the fundus is visible. This membrane has elevations and depressions, with several connective-tissue bands which are generally arranged like radii. These connective-tissue bands often run along the larger blood-vessels, and some of them at times rise from the retina into the vitreous body. The larger retinal blood-vessels usually lie under the membrane. In exceptional cases they appear above it, run for a distance on its surface, and then dip again beneath it. Frequently new-formed blood-vessels are seen in the connective-tissue formation; sometimes, also, shining white spots or points, which are probably produced by regressive metamorphosis. In other cases opacities in the vitreous body, which may appear diffuse, in stripes, or in spots, can be noticed. At times these opacities are membranous, fixed, or movable. Frequently more recent or older hemorrhages can be recognized in the periphery of the retina or in the vitreous body. Blood in the anterior chamber or hemorrhagic discoloration of the iris occurs but rarely.

In this affection the visual disturbance is generally less than what would be expected from the extremely striking and characteristic ophthalmoscopic condition. Sometimes a central scotoma is all that can be found, peripheral vision being moderately well preserved. In other cases the visual field is irregularly contracted, at times but a few unconnected islets in which the patient sees being all that remains. In rarer instances vision is gradually abolished. Vision is destroyed in the cases in which finally amotio retinæ, irido-cyclitic or irido-chorioiditic processes, ectasiæ or progressive atrophy and phthisis are added, these conditions appearing especially when the hemorrhages recur. Light-sense and color-sense, as a rule, remain intact for a long time. Chromopsiæ are of frequent occurrence.

The course of hyperplastic hemorrhagic retinitis is an eminently chronic one. Once developed, it may remain unchanged for years. Even a material improvement has been observed, at least as regards the disturbance of function; especially has this been seen by Manz. In other cases, however, particularly when the hemorrhages frequently recur and when complications such as iritis, irido-cyclitis, irido-chorioiditis, or amotio retinæ exist, the organ may be destroyed by ectasia or cirsophthalmia, though more frequently this occurs by progressive atrophy and phthisis.

The etiological factors producing the condition are unknown. There

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cannot be any doubt that the immediate cause is the hemorrhages into the retina and the vitreous body. What, however, is the cause of these hemorrhages is not sufficiently explained. Heart-disease and affections of the blood-vessel walls have been seen but rarely by Manz and Proebsting, and when present they seemed unimportant. A connection between them and the retinal hemorrhages is, therefore, doubtful. In two cases, one seen by von der Laan and the other by myself, constitutional syphilis was present, but no connection with it could be proved. The same must be stated concerning malaria, which preceded the retinal affection in a case studied by Schulze, although Kries[1] has described hemorrhagic extravasation into the vitreous body after malaria.

The treatment, which in some cases has been of considerable value, consists in the application of inunctions of mercurial and iodine ointments. In the general treatment, inunctions followed by the internal administration of iodide of potassium and Blaud's pills, with injections of pilocarpine, have been employed. Local depletion by means of the Heurteloup leech, and derivatives, in the shape of blisters and foot-baths, have sometimes been tried.

II. RETINITIS; CHORIO-RETINITIS (HYPERPLASTIC TRAUMATIC), RETINITIS SEU CHORIO-RETINITIS (HYPERPLASTICA TRAUMATICA).

Penetrating wounds of the eyeball, through which small foreign bodies, particularly pieces of percussion-caps, have made their entrance (rarely contusion), may produce chronic proliferating processes in the retina or in the retina and chorioid. These may lead to considerable emigration and proliferation of round cells and to the formation of numerous new blood-vessels and granulation-tissue, which, tumor-like, projects into the vitreous body and is changed into connective tissue. Such processes I designate hyperplastic traumatic retinitis or chorio-retinitis. Clinically, the symptom-complex known as amaurotic cat's eye is developed in such cases. In an eye that is generally blind, and at times free from irritation, certain changes may appear. The anterior portion may seem normal. The pupil may be somewhat dilated and the iris but slightly movable. The dioptric media are clear. In such an eye an intense whitish-yellow or reddish-white reflex coming from its depth can be readily recognized with the unassisted eye. With the ophthalmoscope, especially with the direct image, whitish-yellow, tumor-like, nodular masses can be recognized projecting into the vitreous body. In these, neoplastic blood-vessels can be usually observed. Such conditions, when seen in children, may produce a clinical picture like that of glioma endophytum. These are the cases known as pseudo-glioma.

Commonly an error in differential diagnosis is not likely, since there is a clinical history of injury. There are, however, exceptional cases, in which there is no such history, as patients, especially children, desire to conceal any such happening for fear of punishment. Frequently, also, in such

[1] Archiv für Ophthalmologie, Bd. xxiv., 1, S. 157.

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PLATE VIII.

Section of eyeball showing hyperplastic chorio-retinitis.

PLATE IX.

Section showing hyperplastic retinitis with retinal detachment.

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cases we are unable objectively to demonstrate the position of penetration of a foreign body. This is particularly so when a single piece of a percussion-cap, for example, has penetrated into the eyeball through the sclera. In such case we are consulted weeks or months after the accident, by which time the place of entrance can no longer be detected.

Since, furthermore, the tension may be diminished, and in some instances inflammatory processes in the retina and chorioid may be increased, in the very rare cases of true glioma, the making of a correct diagnosis is at times extremely difficult or totally impossible.

If the hyperplastic process is not promptly aborted by enucleation, it generally induces an irido-cyclitis with a tedious course. One synechia develops after another without any symptoms of irritation, and cyclitic products behind the lens interfere with the inspection of the interior of the eyeball. The pupil often becomes occluded, the eyeball grows softer and smaller, and anterior bulbar phthisis (more often painful, complete phthisis bulbi) is developed, and leads to the formation of a painful stump. I have seen a number of such cases, one of which concerned the retina alone and was produced by a contusion from the horn of a cow.[1]

A case of hyperplastic traumatic chorio-retinitis, produced by the penetration of a piece of percussion-cap, I have described.[2] I have reproduced this interesting case on Plate VIII., while Plate IX. shows a case of hyperplastic retinitis with detachment of the retina, which I have also reported.[3]

III. RETINITIS; CHORIO-RETINITIS (HYPERPLASTIC SECONDARY), RETINITIS SEU CHORIO-RETINITIS (HYPERPLASTICA SECUNDARIA).

If such a process of proliferation as above described occurs in the retina or in the retina and the chorioid, after long-continued purulent processes, as in chronic panophthalmitis, or in irido-cyclitic or irido-chorioiditic processes (especially when they have caused a detachment of the retina), I term the condition one of secondary hyperplastic retinitis or chorio-retinitis.

Of this affection we may distinguish two groups:

1. Those which develop from subacute or chronic panophthalmitis, or which appear in the later stages of an acute panophthalmitis. These commonly are of no clinical interest, because the hyperplastic process cannot be clinically demonstrated, since the eye has been previously altered in such a manner that it is impossible to look into the vitreous chamber. Such a secondary or panophthalmitic hyperplastic retinitis or chorio-retinitis cannot clinically be diagnosed from the foregoing, and represents an interesting anatomical condition which may be found accidentally. I have published two such cases and given drawings of them in my paper above mentioned.

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[1] Archiv für Augenheilkunde, Bd. xx. S. 114.

[2] Rozprawy Ceske Akademii, ii. 40.

[3] Archiv für Augenheilkunde, Bd. xxi.

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2. Those which are developed in irido-cyclitis or irido-chorioiditis with amotio retinæ, and in which but rarely the dioptric media are sufficiently clear to permit an observation of the hyperplastic process in the vitreous chamber. Such cases may appear so nearly like glioma that they have repeatedly been described as such. They have also supplied a great number of instances of so-called pseudo-glioma.

XXII. PIGMENTARY RETINITIS (RETINITIS PIGMENTOSA).

Pigmentary degeneration of the retina, or congenital pigmentary retinitis, consists of a pathological alteration of the blood-vessel walls, an insidious proliferation of the supporting tissue of the retina, with atrophy of the nervous elements, and immigration of pigment from the pigment-membrane. On account of the numerous black spots which usually appear in the equatorial region, it generally produces a characteristic ophthalmoscopic picture. These spots, which look like bone-corpuscles, partly cover the blood-vessels and gradually creep towards the posterior pole of the eye. The optic disk appears pallid and is dirty whitish yellow in tint. The blood-vessels of the papilla are attenuated. Subjectively, the disease produces night-blindness. Central visual acuity is preserved for a long time, though the visual field becomes increasingly contracted concentrically. The condition is the result of a congenital binocular disease, which is generally dependent upon consanguinity. As a rule, it forms a much more sharply defined and typical disease-picture than any other retinal affection. Leber[1] has gathered such cases and properly classed them.

During my work I have seen seventy-five cases of this disease, those which I saw as an assistant not being counted, since I have no data about them. Between 1883 and 1893, out of eleven thousand five hundred and fifty-six patients, ten cases of pigmentary retinitis were admitted to my clinic. In my clinical dispensary during the same period I saw thirteen out of forty-seven thousand five hundred and twenty-one patients. In my practice as Royal Provincial Oculist and my private work combined, I have during twenty-six years seen fifty-two cases of pigmentary degeneration of the retina among one hundred and twenty thousand patients.

In this list there were a number of anomalous forms, among which were cases with a typical characteristic ophthalmoscopic picture, but with anomalous subjective symptoms,—namely:

(*a*) Cases in which, although there are no other complications, and the contraction of the visual field is irregular, central visual acuity is considerably reduced from the beginning. This type often shows nystagmus. Such cases are not rare; von Graefe[2] and Donders[3] have mentioned them. Pagenstecher[4] has even gone so far as to separate these cases from

[1] Archiv für Augenheilkunde, Bd. xvii., 1, S. 314.
[2] Ibidem, Bd. ii., 2, S. 283.
[3] Ibidem, Bd. iii., 1, S. 147.
[4] Beobachtungen aus der Augenklinik, Wiesbaden.

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the typical form of pigmentary degeneration of the retina. Leber[1] has described five such cases. (*b*) Cases with characteristic ophthalmoscopic symptoms and normal central vision, but with irregular contracted visual field. Examples of this type have been reported by Mauthner,[2] von Graefe,[3] and Mooren. (*c*) Cases with typical ophthalmoscopic symptoms, normal central vision, and concentric contraction of the visual field, without night-blindness. (*d*) Typical cases in which day-blindness instead of night-blindness is observed. Such a case has been seen by Leber,[4] another by Haase,[5] and a third by de Wecker.[6] I have observed such a case in an hysterical woman.

Another great class is that of cases exhibiting typical disturbance of function associated with anomalous ophthalmoscopic symptoms. This is divided into two types: (*a*) cases in which no pigmentation is found,—such have been seen not infrequently; and (*b*) cases which give the ophthalmoscopic picture of a simple disseminated chorioiditis. Such examples have been described by Mooren and Picard.

A third class is composed of cases with anomalous ophthalmoscopic symptoms and functional disturbances. To these belong the by no means rare types of congenital amblyopia and amaurosis in which the immigration of pigment takes place much later. Leber[7] has observed no less than fifteen examples of this type.

The oldest anatomical illustrations which are probably meant to depict pigmentary degeneration of the retina are given by von Ammon[8] in Figs. 9 and 10 on Plate XIX. There is, however, no statement regarding the history of these eyeballs. The name of "tigered retina," as mentioned by Leber,[9] I cannot find in von Ammon's book. Most of the subsequent anatomical descriptions, which have brought out some important facts, deal either with eyeballs without a previous history, as in von Ammon's case, or with eyeballs that have come from individuals who did not suffer from typical pigmentary degeneration of the retina, but from the secondary pigmentation of the retina which occurs in various eye-affections.

The first anatomical description of cases diagnosticated during life was given by Maes.[10] This was based upon two cases observed by Donders. Maes believed that the retinal pigment came from the chorioid. The best and most exact description is that given by Leber.[11] The case was that of an eye with congenital amaurosis and typical pigmentary degeneration of the retina. He found atrophy of the nervous elements of the retina,

[1] Loco citato.
[2] Ophthalmology.
[3] Loco citato.
[4] Loco citato.
[5] Klinische Monatsblätter für Augenheilkunde, Bd. v. S. 228.
[6] Traité des Maladies des Yeux.
[7] Archiv für Augenheilkunde, Bd. xv., 3, S. 1, and Bd. xvii., 1, S. 325.
[8] Klinische Darstellungen.
[9] Graefe und Saemisch, Handbuch der gesammten Augenheilkunde, Bd. v. S. 35.
[10] Inaugural Dissertation, Utrecht, 1861.
[11] Archiv für Augenheilkunde, Bd. xv., 3, S. 7.

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which was more developed in the outer layers, less so in the fibre-layer, and increasing gradually from the centre towards the periphery. There was hyperplasia of the supporting connective tissue, with the formation of a layer on the inner surface of the fibre-layer. Thickening and sclerosis of the blood-vessel walls, with net-like pigmentation of all the layers, following mostly the course of the blood-vessels, were also present. Prominent changes in the pigment epithelium and numerous excrescences of the lamina vitrea, with small circumscribed exudations undergoing fatty metamorphosis between chorioid and retina, could all be plainly seen.

Later Landolt[1] had the rare opportunity to examine anatomically two eyes in which during life pigmentary degeneration of the retina had been diagnosed. He found almost the same conditions as those noted by Leber,[2] but he concluded that the pigmentary degeneration of the retina is nothing else than a chronic inflammation of the adventitia of the retinal blood-vessels; that is, it is a perivasculitis, which begins at the equator of the globe and gradually creeps towards the posterior pole of the eye.

The walls of the larger arteries are often inflamed and thickened. Their connective tissue becomes hypertrophic, the nervous elements are atrophied by pressure and malnutrition. The process slowly creeps in a centripetal direction, causing the visual field to become concentrically contracted. It also spreads into the deeper parts and to the pigment-layer, especially to those portions which lie directly under the blood-vessels. From here the pigment wanders upward through the channels that have been prepared by the inflammatory process until, once in the retina, it extends and may even proliferate.

The extension of this condition may also explain the formation of excrescences on the lamina vitrea, as well as the circumscribed inflammatory processes found in the chorioid. Furthermore, there is a similar affection of a part of the chorioidal blood-vessels. It also extends inward, leading to hyperplasia of the fibre-layer, with enormous thickening of the limitans interna. Neoplastic connective tissue forms below the fibre-layer and neighboring vitreous body, at which place white round cells, which mostly contain pigment-granules, penetrate into it. Finally, posterior polar cataract, due to the abnormal nutrition of the vitreous body, may develop.

Alt[3] has been fortunate enough to obtain an eye with congenital pigmentary degeneration of the retina which had been enucleated upon account of an intercurrent injury. The conditions found by him were similar to those mentioned. He concludes that in its beginning the pigmentary degeneration is a secondary affection produced by a pathological cell-proliferation (hyperplasia) in the pigment epithelium. He calls attention to the fact that, on account of its function in producing the retinal purple, the pigment

[1] Archiv für Ophthalmologie, Bd. xviii., 1, S. 325.
[2] Loco citato.
[3] Archiv für Augenheilkunde, Bd. vii. S. 376.

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epithelium may be considered as a glandular organ, which may suffer from pathological processes of its own.

Wagenmann[1] has had occasion to examine anatomically a typical case of congenital pigmentary degeneration of the retina. The histological conditions were in the main the same as those described by Leber.[2] Both Wagenmann and Hosch have found cystoid degeneration of the retina in the ciliary region. The former paid especial attention to the conditions of the chorioid, and found its stroma thickened and considerably pigmented. The blood-vessel walls were thickened and sclerosed in many places. The adventitia was opaque, and the intima had proliferated. He believed that the number of blood-vessels was reduced and that the chorio-capillaris was wanting in spots. From these conditions Wagenmann deduced the opinion that the original seat of the affection lies in the chorioid, is due to vascular disturbances, and extends secondarily to the retina. This would not, however, exclude the possibility that the retinal blood-vessels might be attacked at the same time by the same cause.

Deutschmann[3] has been fortunate enough to obtain an eye with congenital pigmentary degeneration of the retina. His results and deductions are very similar to those of Wagenmann.

Certain it is that the number of cases of undoubted typical congenital pigmentary degeneration of the retina which have been examined is as yet too small to justify the drawing of any definite and final conclusions. The opinion of Wedl and Bock,[4] that the immigrated pigment in congenital pigmentary degeneration of the retina always comes from the parenchyma of the chorioid, is not tenable.

Of the many experiments on animals made in order to study the influence of the circulation upon the chorioid and retina, I shall mention only those of Berlin[5] and Wagenmann.[6] The former found, after cutting the optic nerve and the posterior ciliary arteries, a profuse immigration of pigment into the retina from its pigment membrane, this being associated with an atrophy of the nervous elements of the retina. He calls attention to the great analogy between the immigration of pigment in these experiments and in congenital pigmentary degeneration of the retina, and draws attention especially to the fact that in both processes the pigmentation begins at the periphery and that in each the same degeneration of the blood-vessel walls is observed. I must, however, mention in regard to these experiments that, although pigment immigration and atrophy of the nervous elements occurred as in pigmentary degeneration of the retina, there was no hyperplasia of the supporting tissue, and that the rods and cones remained intact,

[1] Archiv für Augenheilkunde, Bd. xxxviii., 1, S. 236.
[2] Loco citato.
[3] Beiträge für Augenheilkunde, Bd. iii., 1891.
[4] Pathologische Anatomie des Auges, S. 203.
[5] Klinische Monatsblätter für Augenheilkunde, Bd. ix. S. 277.
[6] Archiv für Augenheilkunde, Bd. xxxvi., 4, S. 1.

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in contradistinction to pigmentary degeneration of the retina, in which they, as a rule, are quickly destroyed. Wagenmann[1] has shown that after division of the ciliary blood-vessels in rabbits, in consequence of the interruption of the chorioidal circulation, a degeneration in the retina with immigration of pigment into it from the pigment-layer results. He uses these findings of his experiments on animals to explain the origin of the retinal pigment from the pigment membrane, and thus partially supports his opinion of the character of congenital pigmentary degeneration of the retina. He does not, however, consider it correct to take these experimental results as an undoubted answer to the question of the pathogenesis of pigmentary degeneration of the retina, believing that for this explanation further anatomical examination is required.

From all this it is clear that a final decision regarding the true nature of congenital pigmentary degeneration of the retina cannot yet be given, and that a further series of observations must be made.

On account of the pigment, the appearance of the papilla, the changes in the blood-vessels, and the conditions of the chorioid, the ophthalmoscopic picture in typical cases is characteristic.

In typical cases the pigment, which is deep black, appears in great quantity. It is especially accumulated in the equatorial region, and decreases towards the ora serrata and the posterior pole of the eye. As a rule, it follows the course of the blood-vessels, appearing at times in large masses in their bifurcations. Frequently pigment-spots cover portions of the blood-vessels, from which they penetrate to the innermost layers of the retina. These pigment-spots are generally spindle-shaped or appear like corneal corpuscles with numerous offshoots which branch and anastomose with one another. Between them lie spaces that are devoid of pigment. The pigment-spots, which vary in size, are generally of an irregular shape.

The quantity of pigment found in the retina is generally proportionate to the gravity and the duration of the disease-process. Yet there are cases in which the development of pigment is extreme, and others in which it is so slight as to be out of all proportion with the other symptoms, especially visual disturbance. There are even cases of congenital pigmentary degeneration of the retina in which the pigment may be missing, or at least does not develop until many years after birth. In some anomalous types, in which central visual acuity is greatly reduced and which are complicated with nystagmus, large black pigment-spots that are round or irregular in shape are at times found in the macular region. Schweigger[2] has observed such cases.

The papilla appears pale and of a dirty yellowish-white tint, as if covered by a thin mist or veil, presenting the same appearance as it does

[1] Loco citato.

[2] Klinische Beobachtungen.

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in retinitic atrophy. The physiological excavation and lamina cribrosa are not visible. The color of the papilla may sometimes assume several shades from normal: it may be more gray, or more yellow, while sometimes it is somewhat reddish. Always, however, the color is dull and its surface seems dirty. It never assumes the shining white, glossy, like silk, or bluish or grayish (in white light) color of the neuritic atrophy as caused by extra-ocular diseases. In most cases a narrow pale halo surrounds the papilla, its cause being the decolorization of the pigment epithelium. Sometimes this halo appears like the one seen at times in glaucoma.

The blood-vessels are always thin, the arteries being more so than the veins. Sometimes they are accompanied by white lines. In grave and far-advanced cases the blood-vessels seem like red threads and cannot be followed far towards the ora serrata. In the gravest types they may almost disappear, so that it is barely possible to find them. In such cases they look like white bands with pigment margins. At times it may happen that some blood-vessels are still seen as red lines, while others have disappeared, or that one blood-vessel disappears for a varying length and farther on can again be traced as a red thread, thus appearing interrupted.

Changes in the chorioid, which may occur in the most typical cases of pigmentary degeneration of the retina, present themselves ophthalmoscopically as the so-called tessellated fundus, which is due to the dark pigmentation of the intervascular spaces. This, however, is not seen to extend uniformly over the whole eye-ground, some parts appearing lighter than others. At times the larger chorioidal blood-vessels are also found to have white margins, this being caused by sclerosis of their walls. In consequence of the partial decolorization of the pigment epithelium, some pale yellow or yellow-white spots and some brown or black points may be seen. Furthermore, some bright shining spots, that are probably excrescences of the lamina vitrea, may be found.

In typical cases of congenital pigmentary degeneration of the retina a concentric contraction of the visual field is found, while the normal central vision is preserved. There is night-blindness. Central visual acuity remains almost intact for many years, so that it may happen that although the patient can no longer guide himself, yet he can read the finest print. Usually, however, though more slowly, central visual acuity also suffers. In anomalous cases central vision may be considerably reduced from the beginning, thus at times producing nystagmus. The typical condition combined with congenital amblyopia of different degrees or total amaurosis may even be found.

Night-blindness is the most prominent symptom of congenital pigmentary degeneration of the retina. It may exist for years before other functional disturbances are added. Light-sense is diminished, producing so-called torpor retinæ. Such patients, when the illumination is reduced, as in the evening or at night, recognize little or nothing, while with good lamplight or electric light they can see well. Night-blindness is seen in by

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far the greater number of cases of congenital pigmentary degeneration of the retina. In extremely rare instances, instead of night-blindness, day-blindness combined with hyperæsthesia of the retina may be found. The color-sense is usually unaltered, subjective light-symptoms and chromopsiæ being rare.

Concentric contraction of the visual field is one of the most constant symptoms of the congenital type of the disease, and as soon as it has reached a somewhat higher degree it produces such grave visual disturbance that the affected individuals, although able to read the finest print, must be guided when walking in the street. In consequence, the gaze of such people is restless, they being forced, so to speak, to feel the objects with the centre of their visual field as with feelers in order gradually to determine the general picture. With good central vision the diameter of the visual field may sink to from ten degrees to six degrees around the fixation-point. In a few cases the contraction of the visual field is quite irregular, while in rare instances a ring-shaped defect of the visual field, as has been observed by von Graefe,[1] will be found.

Congenital pigmentary degeneration of the retina is frequently combined with other congenital diseases of the eye or of the general organism. Among the eye-affections we meet with congenital diseases, as (ocular) chorioidal, macular, iridic, lenticular coloboma; microphthalmos, irideremia, persistent hyaloid artery (here must be classed Neuffer's case[2] and the one which I reported above), lenticonus, ectopia lentis; stellate posterior polar cataract, persistent capsular artery of the posterior pole of the lens (one case mentioned above from my own practice), pyramidal cataract, and nystagmus.

Acquired diseases, as posterior capsular cataract, posterior cortical cataract, degeneration of the chorioid, may be seen. Mauthner has described a unique case as chorioideremia.[3] In it atrophy of the chorioid of the highest degree combined with pigmentary degeneration of the retina was found. Strabismus is rarely observed.

With regard to affections of the general organism combined with pigmentary degeneration of the retina, deaf-mutism, deafness, and partial deafness are the most frequent. Liebreich[4] has found that five and eight-tenths per cent. of deaf-mutes have pigmentary degeneration of the retina. According to Hoering,[5] idiocy is seen in from twelve to thirteen per cent. Congenital mental weakness comes next, followed by microcephalus (one case reported by Beyer[6] and one by Derigs[7]). Congenital deformities of the most varying kind have been found in the extremities of individuals suffer-

[1] Archiv für Ophthalmologie, Bd. iv., 2, S. 250.
[2] Inaugural Dissertation, Strassburg, 1893.
[3] Berichte des naturwissenschaftlich-medizinischen Vereins in Innsbrück, 2te Th.
[4] Deutsche Klinik, 1861, S. 6.
[5] Klinische Monatsblätter für Augenheilkunde, Bd iii. S. 236.
[6] Inaugural Dissertation, Bonn, 1872. [7] Ibidem, 1882.

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ing from the condition. An extremely rare complication, described by Belarminoff, is glaucoma combined with congenital pigmentary degeneration of the retina.

The course of the disease is slow, taking, as a rule, from thirty to fifty years before blindness results, which usually, however, happens about the fiftieth year of life. Cases have been described in which some remnant of vision has still existed at eighty years. It seems that the anomalous form of this disease in which no pigment immigration takes place has a somewhat better prognosis and must not always be supposed to lead to blindness. This type may become stationary at an earlier period. In newly born children suffering with this disease, pigment in the retina is found in the rarest of cases, generally making its appearance in the first, though more especially in the sixth to the eighth, year of life. Almost without exception it is a binocular affection.

The first visual disturbance is the night-blindness, with its well-known symptoms. While vision in daylight appears normal, sight diminishes rapidly at dusk, so that the patient cannot recognize large objects nor guide himself. After this follows a peripheral contraction of the visual field, which may become more or less marked according to the intensity of illumination. In consequence of the gradual diminution of the visual field, the patient increasingly loses the ability to guide himself. Such subjects continually run into something, and therefore become afraid and uncertain in their movements, even though their central vision may be intact. Later the visual field grows more and more narrow, and finally central vision is gradually diminished until total blindness results. Anomalous cases that deviate more or less from this typical course have been seen.

The real cause of this deleterious process is unknown, and we must be satisfied with the enumeration of the predisposing factors. Liebreich [1] was the first to point out the influence exerted by consanguinity of the parents upon its development. As is known, this plays an important *rôle* in the appearance of deaf-mutism. Liebreich found that in half of the cases consanguinity of the parents could be demonstrated. About the same percentage has been reported by Beyer.[2] More recent observations have shown that this percentage is too high, and have determined it to be between twenty-five and thirty per cent. As pointed out by von Graefe,[3] heredity plays an important part in this disease; one or two generations may escape and the disease then reappear. Often only one among several children of the same parents is affected; the reverse also is possible, as has been observed by Richter [4] and Stievenart.[5] The male sex is more predisposed to it than the female, and the Semitic race more than the Aryan. It is said that it is frequently seen in Turkey, Asia Minor, and the East Indies.

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[1] Deutsche Klinik, 1861, Nr. 6.

[2] Inaugural Dissertation, 1872.

[3] Archiv für Ophthalmologie, Bd. iv., 2, S. 250.

[4] Inaugural Dissertation, Jena, 1828.

[5] Annales d'Oculistique, t. xvii. p. 163.

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In typical cases the diagnosis is easy, and may commonly be made from the statements of the patient before an ophthalmoscope is used. These cases are so well characterized by the congenital appearances, the night-blindness, the concentric limitation of the visual field with intact central vision, by the characteristic ophthalmoscopic picture of the pigment in the periphery of the retina and along the blood-vessels, by the pale veiled papilla and the narrow blood-vessels, and by the mostly diffuse, often punctated decolorization of the pigment epithelium, that the disease cannot be easily confounded with any other.

It is plain from the above detailed course of the disease that treatment is of little value. The most diverse methods have been tried, as depletion, derivants, and resorbents, the constant current, and injections of strychnine, but without success. We must therefore be satisfied with advising the patient to wear smoked or blue glasses in bright light and to save his eyes as much as possible.

ACQUIRED PIGMENTARY DEGENERATION OF THE RETINA (SPECIFIC PIGMENTARY RETINITIS), DEGENERATIO RETINÆ PIGMENTOSA ACQUISITA (RETINITIS PIGMENTOSA SPECIFICA).

It is undeniable that in consequence of syphilis (rarely hereditary) an almost typical form of pigmentary degeneration of the retina may develop. It is probably rare for all the typical symptoms to be found in such cases, and the disease is often monocular, which very seldom happens in the congenital type. Furthermore, central visual acuity is usually disproportionately reduced, and the limitation of the visual field is, as a rule, not perfectly concentric. There are cases, however, which are like the typical ones, and I do not doubt that such forms have been described as typical, especially among the rare anomalous cases. Galezowski goes too far when he claims this etiological factor for all the cases of the disease. Mannhardt and Kugel[1] state that in the East they have frequently seen cases which were undoubtedly developed on a specific basis. It is plain that in such instances an antisyphilitic treatment must be instituted, and it is almost certain that all the cases in which a lasting improvement has followed the exhibition of iodide of potassium belong to this class.

We should also include in this category those cases in which in hot climates pigmentary degeneration of the retina has been stated to be developed by the influence of too bright light. That it is possible under such circumstances, especially on board of ships which have been stationed in tropical countries for long periods of times, and that real epidemics of night-blindness may develop, are well-known facts. Yet it is not proved that the typical pigmentary degeneration of the retina has ever been produced in this manner. Mauthner[2] found the characteristic subjective and

[1] Loco citato.

[2] Loco citato.

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objective symptoms of pigmentary degeneration of the retina in one of the many soldiers who had developed night-blindness in Mexico from long-continued hardships beneath the glowing sun. It is to be regretted that he did not have the opportunity of examining any more of these soldiers.

Secondary pigmentary degeneration of the retina is a term that can be given to cases in which, after more or less protracted severe pathological processes in the uvea and in the external layers of the retina, pigment immigration takes place into the inner layers, and, following the course of the blood-vessels, produces a more or less typical picture of pigmentary degeneration of the retina. In these types the appearance of the blood-vessels and the papilla may be similar to that seen in pigmentary degeneration of the retina, so that it is sometimes very difficult to make a differential diagnosis, which must commonly be based on the anamnesis alone. Two groups can be distinguished.

I. PIGMENTARY CHORIO-RETINITIS (CHORIO-RETINITIS PIGMENTOSA).

In this affection the primary seat of the pathological changes is in the chorioid and the pigment epithelium of the retina, the latter of which partially atrophies and partially proliferates, producing yellow-white and black spots that are visible to the ophthalmoscope. Furthermore, more or less circumscribed exudations, which lead to agglutination and union of these two membranes, are formed between the chorioid and the retina. There is also formation of connective tissue with proliferation of the stroma elements, followed at times by atrophy, changes in the blood-vessel walls, and the formation of excrescences on the lamina vitrea. These were the results of the examinations made by Pope,[1] Förster,[2] Rudnew,[3] and Iwanoff.[4] These changes may be diffuse or may develop in circumscribed foci in the periphery or near the posterior pole of the eye. They are mostly described as a chorioidal affection, since the most important changes take place in the chorioid and in the pigment epithelium, which formerly was considered a part of the chorioid. They are generally classed as disseminated areolar chorioiditis, etc., or, in accordance with Leber,[5] are called external retinitis, or disseminated central and diffuse chorio-retinitis.

Since, however, the pigment epithelium must be considered as a part of the retina, and as serious changes in it, such as softening, atrophy, and proliferation, may be induced as well by retinal as by chorioidal diseases, and, moreover, since it cannot be ascertained by ophthalmoscopic examination, visual disturbance, or anatomical study in the cases belonging to this class, whether the gradual changes in these membranes and the products of inflam-

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[1] Würzburger medicinische Zeitschrift, 1862.

[2] Ophthalmologische Beiträge, 1862.

[3] Archiv für pathologische Anatomie und Physiologie und klinische Medicin, Bd. xlviii. S. 484.

[4] Klinische Monatsblätter für Augenheilkunde, Bd. vii. S. 470.

[5] Graefe und Saemisch, Handbuch der gesammten Augenheilkunde.

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mation and proliferation originally start in the chorioid or in the retina separately or combinedly, it seems best to class in this category those cases in which a secondary pigment immigration into the inner layers of the retina, with other characteristic changes in the retina, occurs, and to place cases in which no distinct changes take place in the retina (especially no pigment immigration) under the head of chorioidal disease.

Etiologically we may in these affections differentiate between cases based on hereditary or acquired syphilis (which represent the large majority), those which develop with anomalies of menstruation at the period of puberty or in the climacteric, and those which occur in progressive myopia with extensive staphyloma scleræ posticum Scarpæ.

II. PIGMENTARY DEGENERATION OF THE RETINA IN DEGENERATE EYES (DEGENERATIO RETINÆ PIGMENTOSA IN OCULO DEGENERATO).

Here are classed the cases in which, after grave pathological processes in the blind and degenerated eyeball, pigment immigration into the retina takes place and is found accidentally during anatomical examination. These cases are of no clinical interest, since they cannot be clinically diagnosed.

This type of cases is found associated with irido-chorioiditis, pupillary occlusion, atrophic and phthisical eyeballs, adherent leucoma, corneal and scleral staphylomata, cirsophthalmos, etc., and often furnishes good material in which to make pathological anatomical researches regarding pigment immigration into the retina.

XXIII. SYPHILITIC RETINITIS (RETINITIS SYPHILITICA).

Until recently there existed a doubt, even among the most prominent ophthalmologists, as to the existence of a disease of the retina independent of the chorioid that is dependent upon syphilis. The existence of such a disease was often denied, although it had been excellently described in 1859 by Jacobson,[1] was seen later by Mooren[2] and Mauthner,[3] and had been pictured by Liebreich.[4] Förster was the most ardent authority in opposing an independent syphilitic retinitis, and described the condition in his classic article[5] as a syphilitic chorioiditis. He maintained his belief in regard to it at a later date.[6] The same position was subsequently taken by Hock[7] and partially by Leber[8] and Schmidt-Rimpler.

On the other hand, syphilitic retinitis has been asserted to occur with

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[1] Königsberger medicinische Jahrbücher, Bd. i. S. 283.

[2] Ophthalmologische Beobachtungen, 1867, S. 287.

[3] Ophthalmoscopie, 1868.

[4] Atlas, 1870.

[5] Archiv für Augenheilkunde, Bd. xx., 1874, S. 33.

[6] Graefe und Saemisch, Handbuch der gesammten Augenheilkunde, Bd. vii., 1877, S. 192.

[7] Wiener Klinik, Bd. ii., 1876, S. 105.

[8] Graefe und Saemisch, Handbuch der gesammten Augenheilkunde, Bd. v., 1877, S. 610.

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great frequency by Ole Bull,[1] having been present in half of all the syphilitic cases seen by him. Schnabel,[2] too, asserts that there is a retinal irritation present in almost all cases of iritis. I can add a partial support to this statement, since in almost every case of severe syphilitic iritis I have seen I have found hyperæmia of the optic papilla, and more rarely neuro-retinitis. Never, however, have I seen a pure retinitis, but rather a chorio-retinitis. In such cases it was almost always possible to prove that the retinitis had been the primary affection to which afterwards the iritis had been added. The existence of a genuine syphilitic retinitis has been placed beyond doubt by the observations of Mauthner, Liebreich, Graefe,[3] and others. Even different forms of this same disease have been described, and we are thus forced to admit the truth of Hirschberg's remark,[4]—namely, that there are many kinds of syphilitic retinitis.

Aside from the pigmentary degeneration of the retina dependent upon syphilis, and the pigmentary chorio-retinitis, the syphilitic chorioiditis so classically described by Förster has thus been acknowledged by most observers as syphilitic chorio-retinitis.

Anatomical researches regarding syphilitic retinitis are very scarce in ophthalmic literature, since, as can be understood, such eyes come under the knife only through an extraordinary accident. The oldest description is by Hutchinson,[5] the microscopical examination of which case was made by Bader. The case was one of hereditary syphilitic chorio-retinitis. Bader found small gray and yellowish foci in the chorioid as well as in the retina, with a thickening of the walls of the retinal blood-vessels. The second examination upon the same subject was made by Edmunds and Brailey.[6] These observers found alterations in the blood-vessels in syphilitic retinitis in both the hereditary and the acquired types that were analogous to those found by Heubner[7] in the cerebral arteries.

The third publication on this subject came from Nettleship,[8] who found in several cases, especially in one of acquired syphilis, the same alterations in the small retinal arteries as had been seen by Edmunds and Brailey. He furthermore determined that the nerve-fibre layer was thickened. The chorioid showed round-cell infiltration in the shape of gummata, and the retina was diffusely infiltrated with round cells that were independent of the chorioidal foci.

The fourth of the published cases is my own,[9] and agrees fully with the

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[1] Nordiskt mediciniskt Arkiv, 1871, Bd. ii. S. 19, and Thesis, Christiania, 1884.
[2] Wiener medizinische Blätter, Nr. 33, 1882.
[3] Archiv für Augenheilkunde, Bd. xii., 2, 1866, S. 211.
[4] Centralblatt für praktische Augenheilkunde, 1886, S. 92.
[5] Royal London Ophthalmic Hospital Reports, vols. i., ii., 1858 to 1860.
[6] Ibidem, vol. x., 1880 to 1882.
[7] Die luëtischen Erkrankungen der Hirnarterien, Leipzig, 1874.
[8] Royal London Ophthalmic Hospital Reports, 1886.
[9] Centralblatt für praktische Augenheilkunde, 1888: Einige Worte über Chorioiditis specifica und tuberculosa.

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results of Nettleship's examination. I have drawn this interesting specimen, which is only incidentally mentioned,[1] on Plates X. and XI. Plate X. shows a sagittal section through the whole eyeball, to allow a study of the pathological changes under a low power. Plate XI. exhibits a highly magnified section through the retina, chorioid, and sclerotic. In the chorioid are found conglomerations of round cells resembling gummata; its blood-vessels appear thickened, especially their intima. Similar changes, although not progressed quite so far, can be seen in the retina. The pigmentary epithelium at places has proliferated and wandered into the chorioid and into the layer of rods and cones, which accordingly shows some defects. Round cells embedded in amorphous material and some round cells containing pigment can be found between the chorioid and the retina. I have also in a case of syphilitic chorio-retinitis seen, aside from a general diffuse infiltration with round cells, aggregations of round cells, like gummata, thickening of the walls of the chorioidal arteries, especially of their intima, and obliteration, with similar conditions in the retina,—although these latter were there less pronounced.

A fifth case has been described by Bach.[2] He found similar changes, especially of the intima, in the smaller arterial branches of the retina, while in the larger ones the adventitia and the intima were altered. These conditions agree with those seen in the former cases, and present the especially important features that the pathological process was limited in this case to the cerebral layer of the retina, and that the chorioid appeared unaltered.

All these anatomical descriptions agree that there are foci of gumma-like aggregations of round cells, with diffuse infiltration of round cells and pathological changes of the walls of the arteries (more often of their intima, sometimes also of their adventitia and media), just as Heubner[3] in his work had proved to exist in cerebral syphilis. According to Heubner, the syphilitic virus at first produces an irritation of the endothelium of the intima, which begins to proliferate, thus narrowing and at times obliterating the calibre of the affected blood-vessel. Later the irritation spreads into the vasa vasorum and produces an inflammation of the adventitia. From here the round cells emigrate into the intima and form a product that is similar to syphilitic granulation-tissue. Still later new blood-vessels may be formed in this newly deposited layer, changing it at times into connective and scar tissue. Caseous degeneration does not occur. Heubner's teachings were rapidly adopted, and found their way into most of the text-books, until Koster[4] and Friedländer[5] showed that the changes found by Heubner in the intima were in no way characteristic of syphilis,

[1] Loco citato.

[2] Archiv für Augenheilkunde, Bd. xviii., 1893, S. 67.

[3] Loco citato.

[4] Vortrag vor der niederrheinischen Gesellschaft für Natur- und Heilkunde zu Bonn, 1875, and Berliner Klinik, 1876.

[5] Centralblatt für die medicinischen Wissenschaften, 1876, S. 65.

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and were to be looked upon simply as examples of typical endarteritis obliterans.

I have repeatedly seen this same type of endarteritis in cases of chorioiditis, tubercular retinitis, nephritic retinitis, and glioma.

From his observations Baumgarten[1] draws the conclusion that in some cases of syphilis a primary disease of the blood-vessels which is not identical with arterio-sclerosis may occur. This affection either bears the anatomical character of a common endarteritis obliterans, or shows its typical origin by the formation of a specific granulation-tissue within the adventitia and media which are supplied by vasa vasorum; to this may be added, subsequently, an anatomically indifferent proliferation of the endothelium.

I must here be allowed to call attention to the extremely exact and excellent work of my former assistant, Professor Deyl,[2] which is of great interest concerning the syphilitic affections of the eye and its adnexa as well as the points of attack of the syphilitic virus in the whole organism. For two reasons this work has not yet found due recognition: first, because it is published in the Bohemian language, and, secondly, because a grave mistake was committed concerning a specimen of syphilitic retinitis which had come from me and which I therefore was forced to correct in my brief communication above.

The work of Professor Deyl, to whom I turned over all syphilitic affections of the eye and its adnexa from my great amount of clinical material, is divided into two parts,—viz., clinical observations and anatomical examinations. Under the head of the former he reports, besides a case of initial sclerosis of the eyelid in a midwife, a series of cases of secondary syphilitic affections of the conjunctiva in adults, children, and the newborn, which he had observed in my clinic, in the clinic for syphilis and dermatology, and in the foundling-house of our university. He found injection, thickening, and dimness of the conjunctiva without secretion, papillary excrescences, and gelatinous swelling.

It was thus ascertained that in nearly all the cases hyperæmia of the small blood-vessels and infiltrations with cells were present. He further discussed the many cases of interstitial keratitis due to hereditary syphilis. He also observed a series of cases of syphilitic iritis, among which he reported two interesting instances from my clinic. Finally, he saw several cases of syphilitic retinitis found in my clinic.

In his studies on the pathological anatomy of the condition, Deyl, on account of the great scarcity of ophthalmic material of this kind, examined the different organs of the body, as follows. 1. Lymphatic glands from individuals suffering from secondary syphilis. These glands were removed from the living. 2. Various organs taken from syphilitic children dead-

[1] Archiv für pathologische Anatomie und Physiologie und für klinische Medicin, Bd. lxxiii., 1878, S. 90.

[2] Ueber die Beziehungen der Syphilis zum Auge.

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born or aborted. 3. The blood-vessels of livers removed from syphilitic bodies. These he studied very particularly. 4. The placentas and umbilical cords of syphilitic children, which yielded results of especial interest, on account of the reversed condition concerning arteries and veins in regard to supply of arterial and venous blood. 5. A specimen of mine, which was wrongly interpreted.

By means of this material taken from the various organs of syphilitic individuals, Deyl studied the behavior of the end-arteries and of the beginning of the lymph-streams of the vasa vasorum, which he also considers as end-arteries, in order to find what part they play in the periarteritis and mesoarteritis of the larger vessels, and of periphlebitis and its part in the dilatation of the peripheral veins and in hemorrhages. He found that in the small end-arteries changes ranging from a simple nuclear swelling of the endothelial cells to obliteration of the vessels take place; that this process in different places is met with in different degrees of intensity; that in the larger arteries the vasa vasorum become affected and produce periarteritis and mesoarteritis, to which secondarily endarteritis is added; that the reduction of the calibre or the obliteration of the blood-vessel may produce hemorrhages; and that the periphlebitis depends on the lymph-stream and produces dilatation of the small veins.

Finally, he surmises that the syphilitic agent selects in an unequal manner as point of attack the region of the small arteries and end-arteries and the beginning of the lymph-stream, and that its deleterious influence may be dependent upon the quantity of oxygen of the blood and its primary transudation. By this he has considerably enlarged the results of the observations made by Koster,[1] Baumgarten,[2] and others, and has proved their correctness as regards diverse organs of the human body and the eye and its adnexa.

Concerning the specimen made by me during my term as assistant, I have to say that I presented it to Deyl, though not the whole eyeball, as Ostwalt[3] erroneously states in his excellent paper, but only one single finished specimen. In this matter a most disagreeable error occurred, which at the time I considered to be the result of a mistake, but which has since been shown to have come about through an act of malice. While I, being then afflicted with an eye-affection, could not examine the specimen thus presented, Deyl was given a specimen from an eyeball with tubercular chorio-retinitis instead of one from an eyeball with syphilitic chorio-retinitis. It will readily be understood why Deyl did not at once recognize this mistake. He knew how familiar I was with the pathological histology of the eye, and had not the slightest reason to doubt my communications to him concerning the specimen. Furthermore, the pathological changes in the chorioid and retina were arranged in a perfectly analogous manner,—that is, the most important changes in the region of the macula

[1] Loco citato. [2] Loco citato. [3] Loco citato.

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in the chorioid, the lesser ones in other parts and in the retina. The pathological changes were, moreover, very similar to those in the syphilitic eyeball. In this case of tubercular chorio-retinitis there were foci, like gumma-nodules, in the chorioid, and in this specimen, also, could be seen changes due to endarteritis obliterans in the arteries of the chorioid as well as of the retina, analogous to those described by Heubner. The fact that in this case giant cells were found in the foci did not contradict its syphilitic nature, since this may happen in syphilitic products, as has been shown by Friedländer[1] and others. A bacteriological examination had not been made, and, of course, could not be made by Deyl on the finished specimen. It must be clear to every one that, under the circumstances, the best histologist might have committed the same error. It is, furthermore, evident that this error does not in the least detract from the results of Deyl's excellent paper, since, on the one hand, the conditions of this particular specimen play a very subordinate part in the many-sided work of Deyl, and, on the other hand, they agree perfectly with the conditions of the real case of syphilitic chorio-retinitis, especially as regards the changes in the blood-vessels, as reported by myself. It is, therefore, a great mistake when Seggel[2] and recently Bach[3] speak of the results of Deyl's work as incorrect. This can be explained only by the fact that these authors were unacquainted with the original of the work, it having been published in the Bohemian language, and that they had read the report in Ostwalt's paper[4] and the necessary correction of it which I had made. Notwithstanding my correction, which concerned only this specimen of the retina, Ostwalt's review is in the main correct, and what is valuable in Deyl's work is not thereby deprived of its intrinsic worth.

(A) SYPHILITIC CHORIO-RETINITIS (CHORIO-RETINITIS SYPHILITICA).

I. *Diffuse Syphilitic Chorio-Retinitis, Diffuse Syphilitic Chorioiditis of Förster* (*Chorio-Retinitis Syphilitica Diffusa, Chorioiditis Syphilitica Diffusa, Förster*).—This typical affection was first described by Förster[5] as chorioiditis. Later it was designated as chorioiditis and as retinitis, until finally the majority of investigators decided in favor of the term chorio-retinitis.

Fine dust-like opacities in the posterior parts of the vitreous body are found in almost all the cases, and are considered pathognomonic. They are hardly recognizable under intense illumination and when the pupil is small. With a plane mirror giving only a weak illumination, and a wide pupil, however, it is possible to see even the finest opacities. These either

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[1] Loco citato.
[2] Deutsches Archiv für klinische Medicin, Bd. xlix., 1889, S. 407.
[3] Archiv für Augenheilkunde, Bd. xviii., 1893, S. 67.
[4] Loco citato.
[5] Archiv für Augenheilkunde, Bd. xx., 1874, 1, S. 33.

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are found uniformly distributed over the posterior parts of the vitreous body, or appear as if arranged in rows and groups. They may be stationary or floating when the eye is moved. Sometimes they develop and increase in number during the course of the disease. Förster has succeeded in noticing how during a few weeks' time the finest dust-like opacities were changed into dense ones, so that they obscured the optic disk. Subsequently they may gradually disappear. Some, however, remain for years after the disease has run its course.

The optic nerve and its neighborhood appear generally as if veiled by these opacities of the vitreous body, and its outlines are indistinct, especially to the nasal side of the disk. The papilla itself is more or less intensely red.

Aside from the veiling by the opacities in the vitreous body, the retina appears uniformly diffusely opaque, particularly in the neighborhood of the papilla and along the larger blood-vessels. The peripheral parts of the vitreous remain transparent. Circumscribed changes are often seen in the fundus, markedly in the region of the macula, where they are seen as bright red, whitish, or gray spots, which sometimes form larger groups. The retinal blood-vessels show no marked changes. The veins are somewhat fuller and the arteries are less distinct than ordinary. Hemorrhages are rarely seen. When the affection ends in atrophy, the papilla appears of a dirty yellowish white.

In consequence of alterations in the pigment epithelium, the background appears speckled. Sometimes the chorioidal vessels and the spaces between them may be recognized. In the retina, particularly in its periphery, immigrated pigment may be seen as in pigmentary degeneration of the retina, yet it does not appear in the typical shape, like corneal corpuscles, but in round pigment-lumps, which do not lie as closely to the blood-vessels as in the degeneration disease. The retinal blood-vessels seem very thin, and are partially invisible.

Central visual acuity is always reduced to from one-sixth to one-hundredth of normal. The visual field nearly always shows considerable defects, especially what is known as paracentral ring-scotoma. Peripheral and star-shaped defects are found, and in grave protracted cases it may happen that vision is retained in small islet-like spots only, the so-called *visus reticulatus*. Scintillating scotomata are of frequent occurrence. The light-sense is reduced, giving the symptom of night-blindness. Photopsiæ and chromopsiæ are almost constant. Micropsia and metamorphopsia are frequent, while the accommodative power is usually reduced.

In about twelve or thirteen per cent. a complication with iritis may be observed. In rare instances the iritis appears first and the chorio-retinitis follows. The latter condition, however, is more often the primary affection, and progresses slowly and gradually, without any pronounced symptoms, to the former.

Diffuse syphilitic chorio-retinitis begins at first very gradually, and its

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course is generally a protracted one. It is prone to relapse, and is seldom cured. When the affection is not grave, when there are no large defects in the visual field, and when rational treatment is instituted early, a cure may be effected. In most cases, however, the disease leaves a more or less reduced visual acuity. The worst result is amblyopia of a high degree with *visus reticulatus,* or amaurosis with immigration of pigment into the retina and retinal atrophy.

The diagnosis is based on the uniform diffuse opaqueness of the centre of the retina and the simultaneous dust-like opacities in the vitreous body. Where the above subjective symptoms are present there can hardly be any difficulty in making a correct diagnosis.

Diffuse syphilitic chorio-retinitis is due to constitutional syphilis. From the ophthalmoscopic picture and the subjective symptoms in such cases syphilis may be diagnosed with the same certainty as kidney-disease can be recognized from the ophthalmoscopic appearances of a nephritic retinitis. It is a late symptom of syphilis, appearing, as a rule, two or three years after the primary infection.

At the same time we frequently find other late symptoms of syphilis; psoriasis, mucous patches, tophi, defluvium capillorum, etc., are frequently present; or these affections may have been cured by preceding antisyphilitic treatment. This affection is relatively frequent at an advanced age, and is especially found among male subjects.

The treatment must be an energetic antisyphilitic one, the best results being obtained by inunctions continued till stomatitis appears. At the same time the patient must remain in a dark room. For after-treatment iodide of potassium and diaphoretics are useful.

II. *Disseminated and Central Circumscribed Syphilitic Chorio-Retinitis* (*Tumid Chorio-Retinitis of Hirschberg*), *Chorio-Retinitis Syphilitica Circumscripta Centralis et Disseminata* (*Chorio-Retinitis Tumida, Hirschberg*).—Under this head belongs the case which I observed in 1865 and published in 1888,[1] and of which I possess anatomical specimens. During my further ophthalmological labors I have seen seven similar examples of the disease, and, as contrasted with diffuse chorio-retinitis, they were mostly found in younger individuals, between thirty and forty years old. Three cases were binocular. Hirschberg[2] has described two such cases as chorio-retinitis tumida. In all there were opacities in the vitreous body, which consisted mostly of fine dust-like bodies. In rarer cases larger flakes could be seen in the posterior parts of the vitreous body.

The optic nerve head is very red, its outlines are dim, and the surrounding area is slightly grayish and opaque. The obscuration of the centre of the retina due to these opacities in the vitreous body is never so marked as in diffuse syphilitic chorio-retinitis. I never saw the papilla

[1] Centralblatt für praktische Augenheilkunde, S. 321.
[2] Ibidem, Bd. xii., 1888, S. 162.

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hidden. In recent cases it is red; in old cases it is dirty yellowish white in tint.

In the majority of my cases the macular region showed important changes. It was somewhat prominent, particularly in the most typical ones. Once I found a larger yellowish-red focus near the optic nerve head. It was from four to five disk-diameters in size, and was raised a little more than one millimetre. It also had an incomplete pigmented margin. Aside from these larger plaques, I saw in several cases, usually in their neighborhood (exceptionally nearer the equator), smaller elevated areas with the appearance at times of dark spots near them. Hirschberg[1] found similar whitish prominent plaques in one case in the macular region, and in another they were situated about five millimetres to the nasal side of the papilla, with two similar foci of a diameter of four or five millimetres and a prominence of one and twenty-hundredths millimetres.

Except some dilatation of the veins, the blood-vessels seem normal. Hemorrhages I have never seen. In older cases the papilla appears of a dirty yellowish white, and the plaques assume a yellowish-white or shiny white color and are no longer prominent. The blood-vessels are very narrow. I have never seen an immigration of pigment, as in the cases of diffuse chorio-retinitis, yet I should not like to maintain that this might not happen, since it may be that I have never seen a case that was old enough for such appearances.

In one eye of a case, which I afterwards dissected, I found the papilla paler than normal and of a dirty yellowish-white tint. In the temporal region between the papilla and macula and covering the latter there was a flat detachment of the retina three disk-diameters in size. Here the retina appeared bluish gray in tint, and was opaque. In the equatorial region I found several smaller yellowish-red plaques of a prominence of about one-half millimetre each. The pigment epithelium in this region and near these plaques was altered. In some places it seemed wanting, while in others it was massed into black spots similar to those seen in simple disseminated chorioiditis. Except in the parts close to the papilla, the retina was not in the least dim. The blood-vessels appeared normal. There were no hemorrhages.

The subjective symptoms are almost identical with those of diffuse syphilitic chorio-retinitis. Yet in those cases in which the important alterations of the fundus affected the macular region, central visual acuity was very much more (and in that minority of the cases in which the affection did not have its seat in the macular region, very much less) reduced than in the cases of diffuse syphilitic chorio-retinitis.

In no instance in which I could make a careful perimetric examination did I find a paracentral ring-scotoma. However, in the majority of the cases I determined the existence of a moderately large central scotoma, and

[1] Loco citato.

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here and there I recognized a round or sector-shaped defect situated in the periphery of the visual field. None of my cases was complicated with iritis, this also being Hirschberg's experience.

The affection runs a slow and tedious course, and inclines to relapses. I have never seen a *restitutio ad integrum;* neither have I seen a case of total blindness with atrophy of the retina. On the contrary, all the cases which I have been able to observe long enough were cured, although in most of them central visual acuity remained considerably reduced.

If the objective and subjective symptoms are well considered, the diagnosis is generally made without difficulty, even without an anamnesis and other concomitant symptoms of secondary syphilis. It might perhaps be confounded with a beginning chorioidal sarcoma or with coalescing tubercles of the chorioid. The objective condition of the optic nerve head and the subjective retinitic symptoms should, however, prevent any error in diagnosis, even when the personal history is wanting or is purposely falsified.

In most of the cases the prognosis must be termed a favorable one, since the ones that I have seen as well as those of Hirschberg all terminated in a relative cure. I do not maintain, however, that worse results are impossible, since the number of cases thus far observed is too small for the deduction of any general rules.

The treatment must be energetically antisyphilitic.

I have briefly reported and drawn the anatomical conditions of one case,[1] which I had been able to examine, with the following results. In the macular region of the chorioid there was a cake-shaped swelling one and three-tenths millimetres high, with longitudinal and transverse diameters of about four millimetres each. Similar smaller and even minute swellings could be seen in the periphery of the chorioid.

Microscopically, the specimen showed that these swellings of the chorioid were foci consisting of densely packed round cells. In the large focus in the macular region I found the results of a regressive metamorphosis of these round cells, and a small amount of pigment. No blood-vessels, however, could be found. The walls of the arteries appeared thickened, especially the intima. The adventitia was less so. The lumen of some of the arteries was narrowed, while that of others was obliterated.

No coarser lesions could be found in the retina, yet the retinal arteries showed the same conditions, which pointed to an endarteritis obliterans. In places they were infiltrated with round cells.

In the left eye I found a small detachment of the retina on the temporal side, beneath which a cake-like swelling, similar to the one in the macular region of the other eye, but smaller in all dimensions, could be seen in the chorioid. In the equatorial region of the chorioid there were similar smaller and larger cake-shaped swellings and foci. The structure of the

[1] Centralblatt für praktische Augenheilkunde, 1888, S. 321.

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chorioidal foci was the same as in the right eye. In the neighborhood of these foci and above them the retinal pigment appeared hyperplastic, while some of its elements had wandered into the chorioid. The blood-vessels showed the same alterations of the adventitia and intima as those of the other eye. The retina in many places was considerably infiltrated with round cells, and its smaller arteries showed the results of endarteritis. There was not any periarteritis. Above the larger foci in the chorioid the rods and cones were wanting or were degenerated. Some colloid excrescences were found upon the lamina vitrea. Amorphous coagulated masses lay between the chorioid and the retina and here and there glued the two membranes together.

(B) SYPHILITIC RETINITIS (RETINITIS SYPHILITICA).

I. *Diffuse Syphilitic Retinitis of Jacobson* (*Retinitis Syphilitica Diffusa, Jacobson*).—It is generally known that Jacobson[1] was the first to direct attention to the fact that the syphilitic process may be primarily located in the retina. He has given an excellent description of this affection.[2] Since his results syphilitic retinitis has been recognized clinically as a separate affection. It is also well known that this retinitis of Jacobson has been opposed and its existence denied. By Förster[3] and his followers it has been described as a syphilitic chorioiditis, while other observers, like Nettleship,[4] take an intermediate position and locate it both in the chorioid and in the retina.

Leber and Michel[5] differentiate between a retinitis of the outer layers, in which the chorioid always participates or even plays the main *rôle*, and a retinitis of the inner layers, in which the chorioid does not participate, or at least does so in a minor degree and much later. This is the genuine primary retinitis.

I differentiate between the above-described diffuse chorio-retinitis and the diffuse syphilitic retinitis of Jacobson. I am well aware of the fact that the conditions described by Jacobson in his diffuse syphilitic retinitis are very similar to those seen in Förster's diffuse syphilitic chorioiditis, and in many points are even identical. I also know that the conditions which Förster advances to prove the chorioidal character of his chorio-retinitis (complication with iritis, night-blindness, opacities of the vitreous body, reduction of accommodative faculty, and alterations in the fundus after the disease has run its course) are not all determinable. I further know that there exist numerous intermediate forms between this form of retinitis and chorio-retinitis. Nevertheless, it is my belief, based on a very large number of cases of syphilitic retinitis and chorio-retinitis which in the course of thirty years I have seen among my patients, that we can assume in a series of similar cases with a certain degree of exactness that the retina, particularly in its inner layers, is the organ first, and often alone,

[1] Loco citato. [2] Loco citato. [3] Loco citato.
[4] Loco citato. [5] Loco citato.

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selected by the syphilitic poison, and that in another series of cases with the same degree of probability we can assume that the chorioid (or perhaps the chorioid with the external layers of the retina) is the part primarily attacked, and that not until later does the process invade the interior layers. There are forms of transition between these two series, as to which it is impossible to decide, and whose classification will depend on the diagnostic tact of the clinician.

The first of these series we should call the diffuse syphilitic retinitis of Jacobson, while the second series are those known as the chorio-retinitis of Förster.

In order to put my opinion on a better foundation, I will here give two anatomical descriptions, the one of my case above mentioned and the other of one recently published by Bach.

In my case the chorioid plays the main *rôle*, and there can be no doubt that the retina suffered only secondarily. Clinically, I have observed a series of cases of gradual transition from my cases of circumscribed syphilitic chorio-retinitis and Förster's diffuse chorio-retinitis, and therefore cannot doubt that if such transitory forms had come under the microscope the changes would have been found to be analogous. Furthermore, I do not doubt that in the typical clinical condition of Förster's retinitis analogous anatomical changes may take place, and yet concerning the participation of the retina and the chorioid in the process the most varying degrees of intensity may be observed.

On the other hand, Bach[1] has described the conditions in a case of relapsing diffuse syphilitic retinitis, in which the pathological changes affected only the cerebral layers of the retina, and has thus brought forward a proof that Jacobson's retinitis exists as a primary disease of the retina, especially of its blood-vessels. Further anatomical examinations will make these points clearer.

Opacities, especially dust-like ones, are nearly always found in the posterior parts of the vitreous body, where they form a light cloud or veil in front of the papilla and its immediate surroundings. They may persist for years after the disease is cured. In rare cases they may be absent: Ostwalt[2] states that in his cases they were more frequently wanting than present. In the posterior pole of the eyeball, independently of the opacities of the vitreous, the retina is opaque. This grayish opacity spreads especially along the blood-vessels towards the equator, where it disappears. In some cases this dimness of the retina is intense in the neighborhood of the papilla, as in Hirschberg's retinitis gyrata albida.[3] Classen[4] and Schweigger[5] have seen a wall-like thickening of the retina around the optic papilla.

Towards the periphery and in the optical centre of the retina small

[1] Loco citato.
[2] Loco citato.
[3] Centralblatt für praktische Augenheilkunde, 1886, S. 92.
[4] Archiv für Augenheilkunde, Bd. x., 2, S. 157.
[5] Augenspiegel, S. 110.

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white foci often appear. Hirschberg has observed their origin and described it.[1] They develop along the blood-vessels, which they sometimes cover like small berries. They are round in form and reddish in tint. Later they become yellowish white, and finally assume a pure white tint. They lie in the retina, and not in the chorioid, as Ostwalt[2] assumes in his cases. In the centre of the retina they are sometimes so minute as to be barely visible.

The blood-vessels show no particular changes, but the arteries are usually thinner and the veins are somewhat thicker than normal. Hemorrhages are extremely rare.

The subjective symptoms do not materially differ from those of diffuse syphilitic chorio-retinitis as Förster[3] has so well described them. The long-continued shimmering of light is especially striking, and becomes extremely disagreeable to the patient. This, as Hirschberg[4] has reported, may sometimes precede for a long period every other visual disturbance, and he explains it correctly as being due to the insufficient supply of arterial blood on account of the affection of the retinal blood-vessels.

In consequence of a reduction of the light-sense, night-blindness and loss of visual acuity are usually very pronounced. Scotomata, especially of the ring-like type, may be found.

The course and the results are similar to those of diffuse syphilitic chorio-retinitis, yet the final chorioidal changes either are wanting or are less marked, so that sometimes we simply find atrophy of the retina with a dirty yellowish-white papilla and very small, partially invisible blood-vessels. Frequently, however, changes in the chorioid also take place. Since in protracted cases the pathological process extends into the outer layers of the retina and into the chorioid, there is immigration of retinal pigment, the reverse of what takes place in diffuse syphilitic chorio-retinitis, in which the affection spreads from the chorioid to the outer retinal layers and afterwards to the inner ones.

The diagnosis and prognosis are similar to those of diffuse syphilitic chorio-retinitis.

This affection sometimes develops in from four to six months after primary infection, as has been observed by Hirschberg.[5] Treatment is the same as that for diffuse syphilitic chorio-retinitis.

II. *Relapsing Syphilitic Central Retinitis* (*Retinitis Centralis Recidivans Syphilitica*).—This is a rare disease of the retina, which was first described by von Graefe[6] in 1866, after de Wecker,[7] Mauthner,[8] and Galezowski[9] had

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[1] Klinische Beobachtungen, 1874.

[2] Loco citato.

[3] Loco citato.

[4] Klinische Beobachtungen, 1874; Beiträge zur Augenheilkunde, i., 1876, iii., 1878; and Berliner klinische Wochenschrift, 1888.

[5] Loco citato.

[6] Archiv für Ophthalmologie, Bd. xii., 2, S. 211.

[7] Loco citato.

[8] Loco citato.

[9] Loco citato.

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pointed out a similar affection, without, however, having related any cases. Schweigger[1] probably referred to the same disease when he spoke of retinitis maculæ luteæ. Mooren's[2] case is probably identical with one of von Graefe's seven cases. A number of examples have been since reported.

Notwithstanding my large practice, I have never treated a case of this type, and therefore cannot speak from my own experience.

Von Graefe, who has seen seven cases, thus describes the affection. Suddenly vision is disturbed. After a few days this disturbance disappears, to reappear and disappear recurrently at short intervals, so that from ten to thirty, or even eighty, relapses may take place. At first vision is good during the intervals, but later the visual acuity becomes reduced. During the attack vision is reduced to a minimum, so that if the affection is binocular it is spoken of as an attack of a periodical blindness.

Ophthalmoscopically a slight dimness is seen in the macula, while the neighborhood of the papilla remains free. The macula becomes gray or grayish yellow, and frequently fine white points appear in groups around it. During the first intervals this dimness disappears, but later it remains stationary. When the relapses have lasted for several years, pigment-spots may develop in its neighborhood, which, however, are not similar to those seen in pigmentary retinitis or chorio-retinitis.

Von Graefe considers this affection as one of the very latest symptoms of syphilis, and uses long-continued or repeated series of inunctions for its relief. Though the affection may resist these for a long time, *restitutio ad integrum* can be reached only in recent cases in which no lasting alterations in the tissues have taken place. A more or less material reduction, especially of the central visual acuity, almost always results. Micropsia has been observed. The affection is often binocular, and may pass over into "diffuse syphilitic retinitis."

In opposition to this description by von Graefe, Reuss has found combined with this affection a papillitis which has resulted in a chorio-retinitis. His and Alexander's cases differ from von Graefe's in the fact that they appeared soon after the infection, in from six to seven months, and in one of Alexander's cases in seventeen months. Otherwise the observations agree with those of von Graefe. It seems, therefore, that this disease may pass over into a "diffuse syphilitic retinitis," or a diffuse chorio-retinitis, or one especially of the circumscribed central syphilitic variety.

III. *Syphilitic Hemorrhagic Retinitis* (*Retinitis Hæmorrhagica Syphilitica*).—Under this head I do not include those exceptional cases in which a diffuse or circumscribed syphilitic chorio-retinitis or a diffuse syphilitic retinitis or a recurrent central syphilitic retinitis is accompanied by scanty hemorrhages, nor those in which a vasculitis of the retina produces an obliteration of blood-vessels with subsequent hemorrhagic infarcts; I mean here only those cases in which in individuals suffering from constitutional

[1] Loco citato.

[2] Ophthalmologische Beobachtungen, 1867.

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syphilis a retinitis or a neuro-retinitis develops sooner or later after the infection (generally a very late one), which from its beginning (I do not mean from its very inception, but from the time when the first ophthalmoscopic examination in consequence of the visual disturbance is made) is characterized by a multitude of hemorrhages. Such affections seem to be very rare; I am at least unable to find many in the literature at my disposal.

I have seen five such cases; I shall, however, relate only the three most typical ones, which I have been able to observe most accurately.

The first, in the wife of a miner, forty-four years old, came to my clinic in 1886. Professor Deyl, who was then my assistant, has, with my consent, already published this case.[1] The patient, who was married the second time, had had a papulous exanthem, had aborted eleven times, had loss of hair, and had suffered from repeated and long-continued, terrible headache. Ten days before her admission she noticed a disturbance of vision in the right eye; this was followed by severe headache and dizziness, during which she became almost completely blind. She was extremely impoverished and miserable. There were cicatrices in the hard and the soft palate, on the genital organs, and about the anus; the cervical glands were swollen; the pupils were small, and the irides reacted very sluggishly.

On ophthalmoscopic examination I found, especially in the right eye, flake-like and dust-like opacities. Both optic nerve heads were very red, and their outlines were indistinct. The retina appeared opaque, but I could not decide how much of this symptom was due to the opacities of the vitreous body and how much of it belonged to the retina itself. In both eyes the arteries were thin, the veins were tortuous and dark, and, especially towards the equator and the periphery, there were innumerable hemorrhages of different sizes and shapes, which lay close to the blood-vessels. In the macular region, in the right eye particularly, there was a large hemorrhage of an irregular oval shape, such as I have sometimes seen in retinal hemorrhages due to menstrual anomalies, and as Liebreich[2] has depicted.

Vision was poor, the patient being barely able to see to count fingers at two metres. I instituted treatment by inunctions, at first carefully, on account of her low condition, but later energetically. She was discharged, considerably improved, at her own wish, and was given iodide of potassium as an after-treatment. After a prolonged absence she appeared in my clinic almost well; yet there were still a few fresh hemorrhages in the retina and some yellowish, marble-like pigmented spots. Her further history I do not know.

The second case was that of the wife of a brewer from the country, seen in my private practice. She had been infected by her husband, and had been treated in the country by several colleagues for various second-

[1] Loco citato.

[2] Loco citato.

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PLATE XII.

Ophthalmoscopic appearance of syphilitic hemorrhagic retinitis.

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ary and tertiary lesions before seeking my aid on account of a disturbance of vision. The patient was thirty-six years old, stout, had aborted twice, and had never conceived since. The visual affection had begun two weeks before her visit, as a central scotoma in the left eye. Externally both eyes appeared normal.

The ophthalmoscopic picture in this left eye was almost exactly the same as that in the right eye of the case reported by Deyl and myself. There were dust-like opacities in the vitreous body, but there were no flakes. The papilla was red, and the retina was opaque, especially at the posterior pole. The retinal vessels looked like those in the preceding case, as did also the hemorrhages, especially the large one in the macular region.

In the right eye I found no opacities in the vitreous body. The papilla was moderately red, and its outlines were slightly indistinct. The retinal arteries were somewhat narrower than normal, and the veins were somewhat hyperæmic. With the perimeter I found in the left eye a large central scotoma with moderately good peripheral vision. Vision with the right eye equalled 6/vi. The ophthalmoscopic picture is given on Plate XII.

I ordered an energetic antisyphilitic treatment. Of the further course of the affection I cannot report anything, since soon after this visit the patient moved into Hungary.

The third case happened in a blacksmith, forty-two years old, who was in my clinic a short time ago. Upon his entrance he admitted that he had been infected years ago and had had secondary symptoms, but he denied this as soon as he found that the sick-fund under such circumstances would not allow him any monetary aid, and thus the personal history remained incomplete. Three weeks before coming to my clinic he noticed a considerable, steadily increasing disturbance of vision in the left eye. No objective signs of syphilis were found, yet the patient complained of a left-sided, severe, and long-continued headache. The right eye was normal. Externally the left eye was also normal. On ophthalmoscopic examination, however, I found dust-like opacities, especially in the posterior parts of the vitreous body of the left eye. The papilla appeared slightly prominent, somewhat red, with hyperæmic veins, and its outlines were indistinct. The remainder of the background was strewn with innumerable hemorrhages of varying size and shape. Some were round or oval, some were striped or flame-like, some were irregular in shape, and some even covered the blood-vessels. The slight dimness of the fundus I attributed to the opacities in the vitreous body. The veins were tortuous, hyperæmic, and dark. The arteries were somewhat smaller, and their walls seemed dim in spots. Here and there the blood-vessels appeared interrupted. Vision equalled the ability to see to count fingers at six metres.

I ordered inunctions, followed by the internal administration of iodide of potassium. No improvement, however, took place; on the contrary, severe, almost unbearable pains appeared in the left side of the head, against

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which antiphlogosis, local bleeding, hydrate of chloral, and subcutaneous injections of morphine were employed in vain.

Later, with normal tension, there appeared pericorneal injection, slight discoloration of the iris, and a small, sluggish pupil. On account of these symptoms atropine was instilled, which, however, produced only a medium and irregular pupillary dilatation. The opacities in the vitreous body increased continually, and soon the eye-ground could hardly be seen, vision being reduced to seeing motions of the hand at one-half metre's distance. The severe pains in the left side of the head resisted all treatment.

Later a few minute erosions appeared on the cornea, such as are seen after herpetic keratitis. The pupil grew somewhat larger; tension increased to T $+\frac{1}{2}$ and $+1$. The vitreous, on account of the numerous opacities, and probably also of the hemorrhages, became dark, rendering the fundus invisible. Eserine could not be borne by the patient, and pilocarpine was substituted, which somewhat alleviated the headache. At his discharge (which, dissatisfied with the result of treatment, the patient demanded, refusing to allow any operative interference), the eye showed considerable episcleral injection, tension was greatly increased, the cornea was steamy, and a small hyphæmia in a shallow anterior chamber could be seen. The iris looked dirty and discolored. The pupil was irregularly dilated, and the iris was devoid of reaction. From the depth of the eye a gray reflex could be obtained. We had here, therefore, a case of syphilitic hemorrhagic retinitis followed by hemorrhagic glaucoma. This form I should designate as malignant syphilitic hemorrhagic retinitis.

The last two cases were analogous to the first two, except that there were no such large hemorrhages in the macular region.

The number of cases which I have mentioned is, I think, too small to allow of any general deductions being made from them. I desire, however, to call attention to the following facts: there was always an extremely large number of hemorrhages over the fundus oculi; the arteries were more or less attenuated, and showed a white dimness in spots; the veins were broad, tortuous, and very dark; the vitreous contained a greater or lesser quantity of opacities; the papilla was red and its outlines were indistinct; and there was always intense and continued headache. Thus there lies before us the picture of a disease which differs from all forms of syphilitic retinitis thus far described, and I would suggest as the etiological factor that a long-continued affection of the blood-vessels, in particular of the arteries, must have preceded the syphilitic hemorrhagic retinitis. It may be that in these cases an endarteritis obliterans was combined with a diffuse syphilitic retinitis.

Schubert has described[1] a partial syphilitic hemorrhagic retinitis. He found a larger hemorrhage, together with a number of smaller ones, in the vicinity of the macula. There were also a few small whitish foci of exu-

[1] Centralblatt für praktische Augenheilkunde, 1881, S. 329.

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dation. The patient suffered from severe headache. There is no statement regarding the condition of the papilla or anything in reference to the presence of opacities in the vitreous body.

Syphilitic Arteritis of the Retina (*Arteritis Syphilitica Retinæ*).—An affection of the retinal arteries analogous to Heubner's affection of the cerebral arteries in cerebral syphilis is recognized ophthalmoscopically when the arteries appear thin and changed into gray or white bands, or eventually cease to be visible, while the veins become enlarged and dark-colored. In this affection hemorrhagic infarcts are here and there seen.

For a long time the visual disturbance may be unimportant, defects in the visual field being especially wanting. Opacities in the vitreous body with marked redness of the papilla and indistinctness of its outlines are not noticeable; neither is night-blindness nor a diminished power of accommodation present. In a word, in most cases no retinal symptoms are observed, the alteration concerning solely the walls of the blood-vessels. The pathological anatomical process is in the main an endarteritis obliterans. Since this process in the retinal arteries occurs, as a rule, simultaneously in the cerebral arteries, we may conclude from the ophthalmoscopic picture that a syphilitic affection of the cerebral arteries is present, and in this manner the ophthalmoscopic examination is of great semeiotic importance for the general practitioner.

All cases of syphilitic arteritis of the retina thus far observed have appeared at a late period of the disease. Schilling[1] was the first to observe the ophthalmoscopic picture of this affection; he, however, erroneously took a white blood-vessel to be a vein. Leber[2] reported another such observation, which was followed by a number of excellent communications upon the subject.

Syphilitic Perivasculitis of the Retina (*Perivasculitis Retinæ Syphilitica*).—Scheffels has described a retinal affection seen by him in a smith, eighteen years old, who suffered from hereditary syphilis. This condition he called perivasculitis or, better, syphilitic periphlebitis of the retina.[3] The papilla appeared, ophthalmoscopically, red; the veins were broad, tortuous, and dark, and surrounded by dark brown-red hemorrhages. The largest hemorrhage lay in the macular region. The arteries were normal. The retina was transparent.

XXIV. PURULENT RETINITIS (RETINITIS PURULENTA).

Until recently an independent purulent retinitis without a combination with chorioiditis was one of the least known of eye-affections, the possibility even of its existence being generally denied.

Thus, Ritter,[4] as the result of experiments on animals, denied the cor-

[1] Medizinisch-chirurgische Rundschau, Bd. xi., 1870, S. 63.
[2] Loco citato, S. 621.
[3] Archiv für Augenheilkunde, Bd. xxii., 1891, S. 374.
[4] Archiv für Ophthalmologie, Bd. viii., 1861, 1, S. 67.

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rectness of all observations concerning purulent retinitis and its possibility. Similarly, basing his conclusion on identical experiments, Schiess[1] stated that wherever purulent retinitis was found it had been preceded by a purulent chorioiditis. Virchow[2] had before this seen a purulent retinitis, yet in his case it was a metastatic affection and the chorioid and the ciliary body were simultaneously affected. I have not the least doubt that in this case Virchow had to deal with a purulent retinitis, although it was said that the independent development of pus in the retina was not proved, and that it could have immigrated into it from the inflamed chorioid. At any rate, it was not an independent retinitis.

The same is true of Nagel's case,[3] in which an operation had been made on the thyroid gland, and of Knapp's three cases,[4] in which puerperal processes preceded it and the inflammation started undoubtedly in the uvea; also of Heilberg's case,[5] where a puerperal endocarditis was present. The important case of Oeller, too,[6] appears, as the author has suggested, to have been of embolic origin, and was combined with pyelitis. The best case of metastatic purulent retinitis in a puerperal subject has been described by Hirschberg.[7] In 1889 I reported a few cases of pure purulent retinitis.

According to my observation, the true independent retinitis occurs in two clinical forms,—as an acute purulent retinitis, with the appearance of a panophthalmitis, and as a chronic purulent retinitis, which while running its course presents the picture of a so-called purulent chronic chorioiditis, the symptom-complex of the so-called amaurotic cat's eye often being the starting-point of the so-called pseudo-glioma, and sometimes being so like a true glioma that a differential diagnosis becomes very difficult, or even impossible.

From their etiology we may easily distinguish between three groups of purulent retinitis: I. Purulent traumatic retinitis. II. Purulent secondary (or induced) retinitis. III. Purulent metastatic (or embolic) retinitis. In every one the purulent retinitis may be acute, exhibiting the form of a panophthalmitis, or it may be chronic, presenting the picture of amaurotic cat's eye. Transitory forms between these two have been observed.

I. TRAUMATIC PURULENT RETINITIS (RETINITIS PURULENTA TRAUMATICA.

Acute Form of Traumatic Purulent Retinitis, presenting the Clinical Picture of Panophthalmitis.—I have seen acute traumatic purulent retinitis most frequently after foreign bodies, especially in cases in which pieces of

[1] Archiv für Ophthalmologie, Bd. ix., 1863, 1, S. 127.

[2] Archiv für pathologische Anatomie und Physiologie und für klinische Medicin, Bd. ix., 1856, S. 317, and Bd. x. S. 175.

[3] Archiv für Augenheilkunde, Bd. vi., 1860, 1, S. 219.

[4] Ibidem, Bd. viii., 1867, 1, S. 127.

[5] Centralblatt für die medicinischen Wissenschaften, 1874, Nr. 36.

[6] Archiv für Augenheilkunde, Bd. viii., 1879, S. 357.

[7] Ibidem, Bd. vi., 1880, 2, S. 299.

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percussion-caps had entered the interior of the eye through the cornea; less frequently when they had passed through the sclera. I have also seen it in perforating wounds, especially through the cornea, though more rarely through the sclera. It has also occurred after severe burns of the cornea and in contusions of an eye which had previously been injured, for instance, when there was an adherent leucoma.

I have carefully studied the connection between acute traumatic purulent retinitis and traumatic panophthalmitis in a long series of panophthalmitic eyeballs from my large collection, and have published the results based upon an examination of twenty-six cases.[1] Since that time I have examined another series (sixteen eyes) with the same affection, and have arrived at the following conclusions:

1. The clinical picture of traumatic panophthalmitis is far from being identical with that of acute purulent chorioiditis, contrary to what thus far seems to have been the general opinion.

2. To the clinical picture of traumatic panophthalmitis corresponds originally as its anatomical basis an acute traumatic purulent retinitis in some cases, and an acute traumatic purulent chorioiditis in others; in some both of these etiological factors have been at work.

3. Acute traumatic purulent retinitis is found to be very much more frequently the original anatomical basis of a panophthalmitis than is a chorioiditis of similar character.

4. Acute traumatic purulent retinitis is found to be the anatomical basis of a panophthalmitis most frequently after injuries or after the penetration of foreign bodies through the cornea which do not injure the uveal tract directly; acute traumatic chorioiditis is found more frequently when the sclera is injured, or when foreign bodies have penetrated through the sclera and directly injured the uveal tract, especially the ciliary body.

The clinical picture, the course, and the results, as well as the prognosis and treatment, of acute traumatic purulent retinitis agree so fully with those of acute traumatic panophthalmitis that we shall here not describe them.

It is commonly impossible to make a positive clinical diagnosis as to whether in a given case of beginning acute traumatic panophthalmitis we have to deal with an acute traumatic purulent retinitis or with a similar chorioiditis. Even a positive anatomical diagnosis is possible only when we have a case in which the enucleation is made early, since in protracted cases the inflammatory process extends to all the tissues of the eye and produces such changes that the origin of the purulent process can no longer be demonstrated.

On Plate XIII. I have given the anatomical conditions of an unequivocal case of independent acute traumatic purulent retinitis from an eyeball which I enucleated four days after a piece of cap had penetrated through the centre of the cornea and lens into the vitreous body. Being

[1] Archiv für Augenheilkunde, Bd. xxi., 1890, S. 348.

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well hardened, this eyeball was made ready for cutting sections after my own method, and put into my collection, to be divided into longitudinal sections years later. The plate shows such a total longitudinal section under a low power, in which the maceration of the tissues and the effects of the consequent reaction can be seen.

Macroscopically, much pus was found in the vitreous body. The retina appeared considerably thickened, and the foreign body lay in the bottom of the vitreous chamber, surrounded by pus and coagulated fibrin. It was also possible for the naked eye to see that the chorioid was not altered.

On microscopic examination I found that the chorioid was normal. The retina, however, was much thickened, especially near the optic nerve head. Here and there a layer of pus and fibrin was seen between the retina and the chorioid. A much thicker layer of fibrin could be seen lying on the inner surface of the retina. The most important retinal changes were found in the nerve-fibre layer. This was enormously thickened, and all its blood-vessels were dilated and gorged with blood; they also showed perivasculitis and endovasculitis. The nerve-fibre layer was infiltrated with round cells, yet the densest infiltration could always be seen close to the blood-vessels, from which its extension in two directions could be traced directly. Whole rows of round cells tending towards the inner surface of the retina, where they formed a direct connection with the cell-layer that covered it, could be recognized. On the other side similar cell-aggregations piercing the outer retinal surface after having wandered through the thickness of this membrane were noticeable. The remaining layers of the retina all exhibited round-cell infiltration. The granular layers were well preserved. The rods and cones were destroyed almost throughout.

From the conditions in this case, to which in the course of years six more have been added, it is clear that the acute traumatic purulent retinitis formed, as an independent affection, the anatomical basis for a beginning panophthalmitis.

Chronic Form of Traumatic Purulent Retinitis, presenting the Symptom-Complex of the Amaurotic Cat's Eye.—This chronic form of traumatic purulent retinitis, the course of which is like that of the so-called chronic purulent chorioiditis, I have seen in only two forms of injury thus far,—*i.e.*, when extremely small foreign bodies, like pieces of caps, had entered the vitreous chamber, and after contusion of a previously injured eye, especially when there was an adherent leucoma.

The clinical appearance, course, results, prognosis, and treatment are the same as those usually mentioned with regard to purulent chorioiditis of a different origin. An undoubted differential clinical diagnosis between the two is impossible.

I have examined a series of such cases, seven in all, and have always found a chronic traumatic purulent retinitis to be the basis of the clinical symptom-complex known as amaurotic cat's eye. In none of these did I

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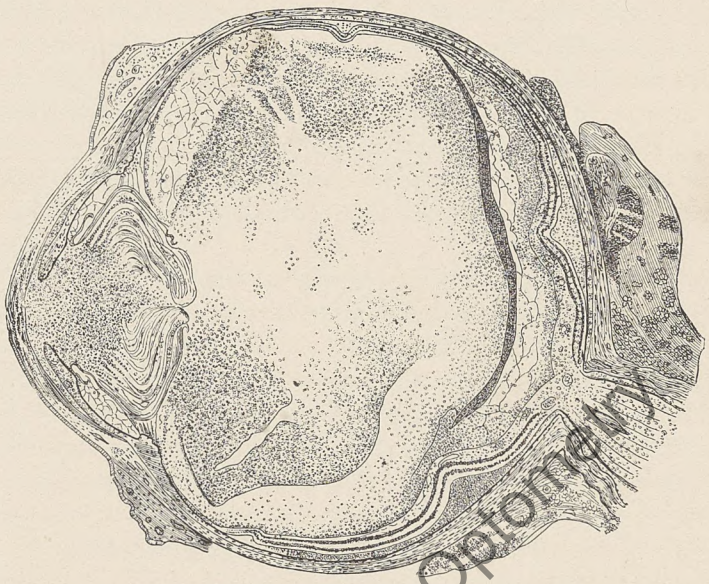
PLATE XIII.



Section of eyeball showing traumatic purulent retinitis.

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find a chorioiditis of a similar character, not even a chorio-retinitis. The chorioid, which was normal in most of the cases, was simply atrophic. Upon the inner surface of the retina in the vitreous chamber I found, in the majority of the cases, a more or less thick shell-like pus deposit, which sometimes filled one-third of the vitreous chamber and sometimes even the whole of it,—pressing the lens and iris forward. The retina was greatly thickened, and full of round cells, as has been described under the acute form. In some cases the retina had been partially destroyed by the purulent process, so that I could find but a few shreds of it in the mass of pus, and these only after a very careful search, as the retina seemed altogether missing. In more protracted cases I found, aside from the pus, products of a hyperplastic retinitis.

These forms of retinitis are interesting not only from an anatomical but also from a bacteriological stand-point. In two cases which I examined when fresh, one a case of contusion of an eye with adherent leucoma, the other an eye into which a small piece of cap had penetrated half a year previously through the cornea and in which the place of entrance could no longer be found, I could not demonstrate any micro-organisms, in spite of the most careful staining. In the majority of the remaining cases I could not make such examinations, the eyes having been preserved for years in my collection.

I can hardly understand how in such a case of contusion of the eye without rupture of its walls these microbes should penetrate to the retina; and yet we have to deal with a veritable purulent retinitis. When a minute piece of cap penetrates into the eye, an accident after which the wound of entrance heals rapidly, leaving no trace, and not until weeks or months afterwards, and very gradually, is pus formed in the retina, I think we can presume that this minute piece, highly heated by the explosion, was aseptic when it entered the eye. It is difficult to comprehend why in such a case the particular organisms should remain for so long a time quiet and finally induce such a mild and slowly progressing inflammation. I therefore think that in such cases, and perhaps also in others, a purulent inflammation is possible in which micro-organisms play no very important part.

II. SECONDARY OR INDUCED PURULENT RETINITIS (RETINITIS PURULENTA SECUNDARIA SEU INDUCTA).

This form of purulent retinitis, which is usually ushered in by the sloughing of the cornea in malignant phlyctenulæ, blennorrhœic, diphtheritic, or serpentic ulcers of the cornea, abscess of the cornea, keratomalacia, etc., more rarely in staphylomata, buphthalmos, cirsophthalmos, etc., and which most frequently leads to acute panophthalmitis and more rarely to chronic inflammatory processes, is commonly of no material clinical importance, since by the original affection an inspection of the interior of such an eye is almost always rendered impossible. The anatomical examination alone leads to the recognition of the nature of the disease.

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In the cases of this form of panophthalmitis of which I had occasion to make an anatomical examination, I have often found that a purulent retinitis, somewhat more rarely a chorioiditis or a chorio-retinitis of a similar character, has been the basis of the panophthalmitic process.

III. METASTATIC OR EMBOLIC PURULENT RETINITIS (RETINITIS PURULENTA METASTATICA SEU EMBOLICA).

It is a well-known but not sufficiently appreciated fact that grave affections of the retina or the chorioid or of both of these membranes may occur during or after severe constitutional diseases of a pyæmic or septicæmic character, and after infectious diseases of varying kinds.

This was earliest known concerning puerperal sepsis, and this eye-affection was described as early as the beginning of this century as puerperal ophthalmia, and by some, such as Jungken,[1] Himly, etc., has been declared to be a milk-metastasis.

Although this affection has been most frequently observed in puerperal sepsis, it may also occur in the varying forms of pyæmia and septicæmia, as after endocarditis, pericarditis, and even pneumonia, in otitis, phlegmon, and after purulent processes in the most diverse parts of the body. It is also seen after periostitis, cerebro-spinal meningitis, exanthematic typhus, Weil's disease, and after variola, scarlatina, etc.

It has been ascertained that the anatomical basis of these eye-affections is a purulent retinitis, a purulent chorioiditis, or a purulent chorio-retinitis, and the septic retinitis of Roth. This form of septic retinitis was first described by Roth[2] in an exhaustive paper, and contrasted with embolic panophthalmitis as a disease of the retina *sui generis*, analogous to nephritic retinitis, etc. Litten[3] in his first publication agreed with Roth, but changed his opinion later, and, like Leber,[4] Kahler,[5] and Wagenmann,[6] confounded it with embolic panophthalmitis. More recently Herrnheiser[7] has advocated Roth's opinion.

During my service as assistant in the eye clinic at Prague and during the years 1860–70 I examined a great many cases of this form of septic retinitis ophthalmoscopically, and I have in my possession slides of three such cases. This experience leads me to agree with the opinions of Roth and Herrnheiser concerning this affection. Therefore I do not classify this form of retinitis among the purulent varieties, and I shall speak of it, together with analogous affections, later.

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[1] Augenheilkunde, 1842.

[2] Deutsche Zeitschrift für Chirurgie, 1872, Bd. i. S. 471.

[3] Charité-Annalen, iii.; Berliner klinische Wochenschrift, 1878; Zeitschrift für klinische Medicin, 1881.

[4] Graefe und Saemisch, Handbuch der gesammten Heilkunde, Bd. v. S. 564.

[5] Zeitschrift für Heilkunde, 1880.

[6] Archiv für Ophthalmologie, 1877, Bd. xxx., 2, S. 147.

[7] Klinische Monatsblätter für Augenheilkunde, 1892; Zeitschrift für Heilkunde, 1893.

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Metastatic purulent retinitis may appear in an acute form with the picture of panophthalmitis, or it may assume a chronic form with the symptom-complex of amaurotic cat's eye. Between the two there are many varieties of transition.

(B) ACUTE METASTATIC RETINITIS, METASTATIC OR EMBOLIC PANOPHTHALMITIS (RETINITIS METASTATICA ACUTA, PANOPHTHALMITIS METASTATICA SEU EMBOLICA).

This affection was described in the first decades of this century by Tenon, Jungken, Himly,[1] and others as puerperal ophthalmia. Fischer,[2] Arlt,[3] Hasner,[4] Pilz[5] (all of the Prague School), have recognized this disease and have described it at length. Then came the epoch-making publications by Virchow[6] and H. Müller,[7] which furnished important anatomical data; also the important publications by Nagel and Knapp. Heiberg[8] was the first to demonstrate micro-organisms in the chorioid as well as in the retina in a case of metastatic chorio-retinitis. Then followed the important observations of Roth.[9] Leber[10] lays particular stress on the fact that the cell-infiltration always begins in the nerve-fibre layer and spreads thence into the other layers and into the vitreous body. This I found to be so in all the recent cases I could examine. In an instance seen by Beck,[11] in which metastatic panophthalmitis followed a pleuro-pneumonia, the retina was dissolved in pus, and the chorioid was but moderately infiltrated. Emboli could not be found. Kahler[12] has reported an example of septic retinitis of Roth and one of metastatic chorio-retinitis, and has endeavored to show that the two processes are alike. Litten[13] reports a series of cases of septic retinitis of Roth, as well as some of metastatic panophthalmitis, which he now considers to be identical, while previously he had looked upon them as two different processes. In some of these cases he demonstrated the presence of micro-organisms.

Hosch[14] found the retina much more affected than the chorioid, and discovered the presence of what he considered to be leptothrix. In a case of binocular metastatic panophthalmitis in a puerperal subject Wagenmann[15] found multiple emboli of streptococci. Mitvalsky has examined two cases from puerperal subjects from my clinic and has based an article on them. In an exhaustive paper Herrnheiser[16] has reported seven cases of septic retinitis and metastatic panophthalmitis seen in the German eye clinic of Prague. In the cases of metastatic panophthalmitis he found the staphylococcus pyogenes aureus, the streptococcus pyogenes, and a new diplococcus.

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[1] Loco citato. [2] Lehrbuch der Augenheilkunde, 1846.
[3] Ibidem, 1853. [4] Entwickelung der anatomische Bericht des Auges, 1847.
[5] Lehrbuch der Augenheilkunde, 1859.
[6] Loco citato, 1856. [7] Gesammte Schriften, 1856.
[8] Loco citato. [9] Loco citato. [10] Loco citato.
[11] Memorabilien, 1877. [12] Zeitschrift für Heilkunde, 1880.
[13] Berliner klinische Wochenschrift, 1881. [14] Von Graefe's Archiv, xxii., 1880.
[15] Loco citato. [16] Loco citato.

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The differences between septic retinitis and metastatic panophthalmitis he maintains decidedly.

I have at my own disposal concerning this eye-affection experiences as numerous as they are sad, together with remembrances dating from 1850 to 1870, especially those years in which I served in the obstetric clinic, and later as an assistant in the eye clinic.

In those days puerperal processes were common in the public institutions as well as in private practice. I remember several days during which I saw daily more than twenty individuals suffering from puerperal fever, of whom four or five had metastatic eye-disease. The number of cases which I examined in those days ophthalmoscopically was enormous; I could not give it to-day, since in the war year of 1866 nearly all my papers were lost. I know, however, that it largely exceeded one hundred. In those days I collected a great deal of anatomical material, and this was the easier since I was then in all Bohemia the only one who studied pathological histology, when even normal histology was unknown to almost all of my colleagues. Since we were on the most friendly terms, and because of the vast and undivided material of our University, almost all such cases reached my hands. Aside from numerous eyeballs which I cut into sections in those days, about sixteen or seventeen well-prepared but intact ones with metastatic panophthalmitis remained, which, together with other interesting specimens, disappeared only a few years ago. Subsequently, on account of antisepsis and asepsis, such cases grew increasingly rarer, and thus it happens that I have not seen a case in my clinic for years. Two cases from my clinic I have described briefly in my article "Beiträge zur pathologische Anatomie der Panophthalmitis."[1] I have not there mentioned those cases in which the metastatic panophthalmitis was due to a purulent chorioiditis or a purulent chorio-retinitis, and in which the retinal changes played an unimportant part.

Concerning the connection between acute purulent metastatic retinitis and metastatic panophthalmitis, I found, as in traumatic panophthalmitis, that a retinitis is much more frequently the initial change than a chorioiditis or a chorio-retinitis of similar character. The same conclusions have been arrived at by Herrnheiser[2] from his own observations and a study of the literature on this subject.

The clinical course and the results, if death does not overtake the patient, are the same in metastatic panophthalmitis as in traumatic panophthalmitis. We observe the same chemotic swelling of the reddened and heated lids and the bulbar conjunctiva, the same protrusion and immobility of the eyeball, and the same whitish-yellow reflex from the eye which has rapidly grown blind. The pain may be just as severe, yet, from clinical observation, I may state that in metastatic panophthalmitis,

[1] Archiv für Augenheilkunde, Bd. xxi. S. 373.

[2] Loco citato.

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in spite of the turbulent appearance of all the symptoms, I have seen a more indolent and painless course than in traumatic panophthalmitis. Intermediate forms which tend more to a chronic and milder course than in traumatic panophthalmitis also occur. A differential diagnosis cannot easily be made, whether in a particular case one is dealing with a metastatic panophthalmitis that is due to retinitis or with a chorioiditis or purulent chorio-retinitis. In the later stages such a clinical distinction is impossible. In fresh cases I have never made a mistake, when the vitreous body was not densely infiltrated with pus and when some, however slight, details of the fundus could be seen.

The anatomical conditions also are analogous to those described in traumatic panophthalmitis. I have always found the first changes in the nerve-fibre layer, where the cell-infiltration was the greatest, and especially near the blood-vessels, which were affected by perivasculitis and endovasculitis. The remaining parts of the eyeball (vitreous body, lens, chorioid, cornea, sclera, and Tenon's capsule) may show similar symptoms in a varying degree, as has been found in traumatic panophthalmitis. In metastatic panophthalmitis, however, the bacteriological conditions are of special importance.

After the observations published by Heiberg, Roth, Litten, Kahler, Hosch, Hirschberg, Wagenmann, Herrnheiser, and others, it is generally accepted that micro-organisms which penetrate into the eye as emboli must be considered as the etiological factors of metastatic panophthalmitis. By the majority of these observers the microbes have been most frequently demonstrated as more or less bulky emboli situated in the blood-vessels of the nerve-fibre layer, in the pigment epithelium, and in the vitreous body.

METASTATIC OR PROPAGATED CHRONIC PURULENT RETINITIS (RETINITIS PURULENTA CHRONICA METASTATICA SEU PROPAGATA).

Metastatic purulent retinitis, especially that of puerperal origin, may run a slow and quiet course and present the picture of the commonly so-called chronic purulent chorioiditis with the symptom-complex of amaurotic cat's eye.

It is, therefore, decidedly an error when Roth distinguishes his septic retinitis from the grave embolic panophthalmitis simply as a slighter affection of the fundus oculi, and when, later, Litten, Kahler,[1] and others declare this form of septic retinitis to be a milder variety of metastatic panophthalmitis, since a true mild form of metastatic purulent retinitis exists which runs its course without grave symptoms of irritation, and which alone can be distinguished from the acute form. Such a chronic purulent retinitis or chorio-retinitis or chorioiditis may be eventually observed, and is not at all rare, especially during puerperal processes. This affection has, further, been

[1] Loco citato.

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observed after infectious diseases, as scarlatina, variola, etc. A series of cases have been seen during and after epidemic cerebro-spinal meningitis and in other forms of meningitis. It has not been ascertained whether such cases have been produced by propagation from the meninges by way of the lymph-channels of the optic nerve, or whether they have been of metastatic origin. In explanation, however, most observers accept both ways of infection. There are also some rare cases in which, in spite of a thorough clinical history, no etiological factor can be demonstrated, and which, since they generally occur in children, may be mistaken for glioma.

I have observed a great many cases clinically after cerebro-spinal meningitis, especially during my service as assistant, and I am convinced that in most if not all of them the condition was one of chronic purulent retinitis, since I believe a chorioiditis of a similar character is extremely rare. I have examined anatomically five cases in which I have found a chronic purulent retinitis to be the basis of the process, and have not seen an analogous chorioiditis in a single instance. The vast majority of such cases which I have studied clinically occurred in puerperal subjects and in children convalescing from acute infectious diseases. Once I saw a case in an adult after typhus fever; I also have found two cases after purulent meningitis. I have never seen a case occur after epidemic cerebro-spinal meningitis.

The clinical symptoms, course, and results are analogous to those of chronic traumatic purulent retinitis. The eye is free from irritation or shows but a slight episcleral injection. Rarely a slight œdematous swelling or a serous chemosis of the bulbar conjunctiva will be found. At first the dioptric media are clear and the eye presents the symptom-complex of amaurotic cat's eye. Usually there is no pain. When such cases occur early in childhood and the history is doubtful or missing, they may be mistaken for glioma of the retina, constituting the cases of so-called pseudo-glioma.

For the differential diagnosis the shell-like concave form of the fundus is especially valuable, as well as the absence of new-formed blood-vessels and the generally reduced tension of the eyeball. When, however, in exceptional cases intra-ocular tension appears increased, or when in protracted cases, aside from the purulent process, hyperplastic inflammations occur, or when the fundus presents a nodular appearance and new-formed blood-vessels can be seen, a differential diagnosis becomes impossible.

Later the anterior chamber grows more shallow, the pupil becomes somewhat dilated, the iris is slightly discolored and reacts sluggishly, posterior synechiæ successively develop without any special symptoms of irritation, and the whitish-yellow growths in the vitreous chamber gradually but steadily advance towards the posterior pole of the lens. The pupillary edge becomes glued to the anterior lens-capsule, and the lens grows dim, rendering an inspection of the interior of the eye impossible. The eyeball gradually grows softer and smaller, and progressive bulbar atrophy which

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may lead to either anterior or complete phthisis bulbi develops. Intermediate varieties between this chronic form and the rapid metastatic panophthalmitis are often seen, and acute exacerbations may at intervals appear in the chronic form. In rare cases the sclera becomes perforated.

The chorioid either is slightly infiltrated with round cells and slightly swollen in places or is normal. In some types I have found this coat atrophied. The retina is greatly swollen and thickly infiltrated with round cells, pus, and fibrin, as is somewhat more frequently found in the acute variety. From the margin of the optic nerve head to the ora serrata the membrane is changed into a mass of pus, in which only single shreds of retinal tissue densely infiltrated with pus-cells and remnants of blood-vessels can be with difficulty demonstrated. The optic nerve head is sometimes excavated and the nerve itself filled with round cells. In the vitreous chamber fibrin and pus accumulations varying in size can be found. Sometimes the vitreous humor as far forward as the posterior pole of the lens, which is pressed forward, becomes a mass of pus. The conditions of the lens, the ciliary body, the iris, and the cornea vary.

In slowly progressing cases hyperplastic inflammation may take place. This may be partial or diffuse, and produce granulation masses, connective material, and scar tissue.

XXV. SEPTIC RETINITIS OF ROTH (RETINITIS SEPTICA—ROTH).

Roth has reported that during pyæmic and septicæmic processes he has seen a retinitis which greatly resembled albuminuric retinitis or the one found in pernicious anæmia, and which had nothing in common with purulent metastatic retinitis. He compares the condition with panophthalmitis, though he states that it is more frequent and that it runs a more favorable course. He says that it is not due to embolism, but that it must be considered as dependent upon chemical change in the blood and alterations in the blood-vessel walls. Niederhauser[1] agrees with Roth. Leber[2] considers the condition to be a milder form and a forerunner of the embolic types of disease due to micro-organisms. Kahler and Litten[3] consider the condition identical with embolic or metastatic processes. Bayer[4] takes the same view as Roth. Personally I have seen a great many cases of septic retinitis ophthalmoscopically and have examined some of them anatomically, and I am convinced of the correctness of Roth's and Herrnheiser's positions. The majority of the cases seen by me, however, did not occur during puerperal fever, but were found in other septic affections.

Ordinarily the exterior of the eye appears unirritated. In rare cases there is a slight œdema of the conjunctiva, with small hemorrhages in the conjunctival tissue. The iris is normal and reacts well. The refractive

[1] Inaugural Dissertation, 1875.
[2] Loco citato.
[3] Loco citato.
[4] Tageblatt der 58. Naturforscher Vereiner, Strassburg, 1885.

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media are clear. The optic disk is neither red nor sharply defined. In the region of the optic nerve head and the macula, hemorrhages varying in number, shape, and size, and sometimes having white central spots, can be found. In this situation there are also white spots (so-called Roth's spots) varying in number, shape, and size, and similar to those seen in albuminuric retinitis. These spots appear later in the course of the disease than the hemorrhages. Hemorrhagic extravasations on the papilla are of rare occurrence. There is no ciliary irritation. The course of the affection is painless and slow. Visual acuity is not materially diminished, except in rare instances in which a hemorrhage lies in the macular region, in which case central vision suffers materially, giving rise to central scotomata.

Prognosis is favorable, although traces of hemorrhages and sometimes hæmatogenous pigment may be observed for a long time. The appearance of a septic retinitis does not affect the general prognosis. It may or may not be seen in the gravest and in the lightest forms. It cannot serve, therefore, as a semeiotic sign for the graver cases or even for the possibility of death of the subject.

Upon anatomical examination it will be found that the hemorrhages usually lie in the nerve-fibre layer, especially near the papilla. There are no material changes in the blood-vessel walls. The white spots which are sometimes observed in the centre or at the margin of the hemorrhages, and at times appear to be independent of them, will be found to consist of hypertrophic nerve-fibres. At times hæmatogenous pigment is found. Leber, Litten, and Kahler have seen micro-organisms, while Roth and Herrnheiser deny their presence.

In conclusion, I may say that inflammatory symptoms have not been found with septic retinitis either clinically or anatomically. On this account, Herrnheiser does not consider it correct to term it a true retinitis, and proposes the name "retinal affection, or retinal changes due to sepsis." I think that, in analogy with other affections, it might be termed septic hemorrhages in the retina.

XXVI. LEUKÆMIC RETINITIS, LEUKÆMIC NEURO-RETINITIS (RETINITIS LEUKÆMICA, NEURO-RETINITIS LEUKÆMICA).

In 1861 Liebreich was the first to describe and picture a case of retinitis due to leukæmia. Becker has reported two cases and has given a picture of two different stages of one of them. Saemisch[1] has seen a case and has reported the results of an anatomical examination.

The right eye presented hemorrhagic glaucoma, the left one a retinitis with a great many hemorrhages, so that it was interpreted as an apoplectic retinitis. The chorioid was thought to be an important factor. Leber[2] reports extensively on the anatomical examination of the eyes of a leukæmic

[1] Zeitschrift für medicinische Blätter, Bd. vii., 1869.
[2] Ibidem.

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patient. Besides white spots, he found numerous small hemorrhages, especially in the periphery of the retina, there increasingly lessening towards the papilla. The white foci were somewhat prominent and round. Leber declared them to be small lymphomata, such as are seen in other localities. The papilla and retina were slightly opaque; the outlines of the former were indistinct. No fatty degeneration, sclerosed nerve-fibres, or pigmentary changes could be determined, while no alterations in the chorioid, except a hyperæmia, could be seen.

Roth[1] reports on the anatomical examination of a case of leukæmia. He found numerous small hemorrhages in the periphery, lymphoid infiltration in the blood-vessel walls, and, contrary to Leber, fatty degeneration and sclerosed nerve-fibres. The hemorrhages appeared of the normal red color. Schirmer, who ophthalmoscopically examined four leukæmic patients, found similar appearances to those that have been depicted by Liebreich.

Reincke[2] reports the anatomical conditions seen in a case of leukæmia. From what he saw, he opposes Leber's opinion that the white plaques must be considered lymphomata, and agrees with Saemisch, who declared them to be hemorrhages with the white blood-cells placed in the centre and surrounded by the red ones. The absence of a reticulum and the sharp definition of the plaques militate against Leber's view. Reincke also opposes Saemisch's conclusion that the chorioidal affection is the prime factor in this disease. He does not consider so-called leukæmic retinitis an affection that is characteristic of leukæmia, but believes that it consists in a series of hemorrhages just as they occur in the retina without leukæmia, the ophthalmoscopic picture being altered by the leukæmic character of the blood itself.

In one case Perrin[3] found the optic nerve head and the retina as far forward as its equator swollen and milky opaque. The outlines of the disk and the contours of the blood-vessels were veiled. The veins were dilated, dark blue, tortuous, and seemed to be pulsating violently (?). Numerous hemorrhages in stripes with white centres could be seen. From these conditions he doubts whether there is a retinitis that is characteristic of leukæmia, and thinks that we should consider leukæmic and albuminuric retinitis identical, especially since leukæmia and albuminuria may coexist. It seems to us, however, that it is rather rash to draw such conclusions from the observation of one case. Poncet,[4] in examining this case anatomically, found numerous hemorrhages with white centres which consisted of an aggregation of white blood-corpuscles; these findings he considers characteristic of leukæmic retinitis. The papilla was projecting mushroom-like into the vitreous. The chorioidal vessels were filled with white blood-cells. No trace of changes such as are seen in albuminuric retinitis could be found.

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[1] Archiv für pathologische Anatomie und Physiologie und für klinische Medicin, Bd. xlix., 1870.

[2] Ibidem, Bd. li., 1870.

[3] Gazette des Hôpitaux, 1874.

[4] Gazette médicale de Paris, 1874.

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Poncet explains what he saw in leukæmic retinitis by assuming three stages of the affection: in the first the blood-vessels are filled with white blood-cells; in the second numerous hemorrhages take place; and in the third small leukæmic tumors may be formed.

Burk[1] reports a case of leukæmia in which the ophthalmoscope revealed only symptoms of vascular stasis with a yellowish color of the fundus. Anatomically, an almost complete obliteration of the superior ophthalmic vein could be seen.

Michel,[2] while examining a case which had not been observed ophthalmoscopically during life, found considerable stasis-hyperæmia in the retina and numerous hemorrhages, the cause of which he determined to be a complete obliteration of the central retinal vein. This fact, together with Burk's experience, led Michel to explain leukæmic retinitis to be simply the consequence of an obstacle in the venous blood-stream. In the same manner he endeavored to explain Leber's case of leukæmic tumors in the eyelids, bilateral exophthalmos, and hemorrhagic retinitis.

It seems to us that it is imprudent to generalize such an explanation of leukæmic retinitis, and, since this retinitis is always bilateral, it is at least improbable that an obliteration of both central veins of the retina or the superior ophthalmic veins should have occurred in all cases. It is much more natural to assume that in leukæmic individuals sometimes such a hemorrhagic retinitis due to obstacles to the venous blood-stream may be observed aside from the typical leukæmic form of retinitis.

Deutschmann[3] saw during a hurriedly made ophthalmoscopic examination in a case of rapidly fatal leukæmia a pale color of the fundus, dilated blood-vessels, and an almost normal optic nerve head. He also found numerous hemorrhages with white centres and a red halo, and a large white spot without a red halo. The anatomical examination agreed with the ophthalmoscopic picture. There were hemorrhages, consisting of red blood-cells and foci of white blood-cells surrounded by red ones, in all the layers of the retina. The white spot without red halo consisted of sclerosed nerve-fibres.

Oeller[4] reports what he found in a case in which no ophthalmoscopic examination had been made. The papilla and the retina around it were considerably swollen and dim. There were numerous hemorrhages, chiefly in the nerve-fibre layer, especially around the optic disk. Some of them entered the deeper retinal layers. In the former the red blood-cells predominated, while in the others which lay close to the blood-vessels white blood-cells prevailed. Few only had a red border. The optic nerve head was swollen, and was filled with nuclei. The chorioid was hyperæmic, and many white blood-cells could be seen in the full blood-vessels.

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[1] Inaugural Dissertation, Erlangen, 1876.

[2] Deutsches Archiv fur klinische Medicin, Bd. xxii., 1878.

[3] Zeitschrift für klinisch-medicinische Blätter, Bd. xvi., 1878.

[4] Archiv für Augenheilkunde, Bd. xxiv., 1878.

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Deutschmann[1] has reported three cases of leukæmic retinitis, one in acute, another in subacute, and a third in chronic leukæmia. He gives a characteristic ophthalmoscopic picture of the first two and an anatomical examination of all three. In the first the hemorrhages predominated in the ophthalmoscopic picture. They then disappeared, leaving dark pigment spots and stripes, after which new ones appeared. The papilla was pale and indistinctly outlined. In the second case the dilatation and tortuosity of the veins were greater than in the first. There were hemorrhages, and in the periphery there were white plaques. In the first case the fundus was pale red, and in the second it was light yellow in tint. Anatomically he found swelling of the papilla and the surrounding retina. The blood-vessel walls were infiltrated with round cells. The connective tissue of the retina was hypertrophied and thickened, so that the granular layers appeared removed from one another. There were numerous hemorrhages and round cells in the nerve-fibre layer. In places the blood-vessels were ectatic, filled with white blood-cells, and projected towards the vitreous humor and the chorioid. There were round patches of sclerosed nerve-fibres. The chorioidal blood-vessels were full of leucocytes, and the stroma was diffusely infiltrated with round cells. He could find no micro-organisms.

In 1870 I saw a characteristic case of leukæmic retinitis in my private practice in a man, thirty-six years old, who was suffering from subacute spleno-lymphatic leukæmia. Externally both eyes were normal. The media were clear. The fundus appeared pale orange-yellow in tint, paler in the right eye than in the left. Each optic nerve head was swollen and opaque, and its outlines were invisible. It protruded mushroom-like into the vitreous humor, as in genuine papillitis, and upon it numerous hemorrhages in stripes, in association with white stripes and spots, could be seen. The retina at the posterior pole, especially around the papilla, was slightly opaque. Numerous minute hemorrhages, shaped like stripes, flames, and points, were situated in the retina. In the macular region and towards the periphery there were numerous round or elliptical, small, dull-white plaques, most of which projected over the retinal surface and were surrounded by a red border of various widths. The veins were extremely dilated and very tortuous; the arteries were less so. Both kinds of blood-vessels were pallid and in places had white borders.

The diagnosis of binocular leukæmic papillo-retinitis was made. Unfortunately, however, this was the only occasion I had to examine this patient ophthalmoscopically, as soon after he died. It was impossible for me to secure the eyes for microscopical examination. The accompanying plate shows the picture of this case, made from my sketch.

On the basis of cases from literature and my own observations, the affections of the retina in this disease can be separated into two groups:

[1] Beiträge zur Augenheilkunde, Bd. iv., 1892.

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(1) typical leukæmic retinitis, or papillo-retinitis; and (2) retinal hemorrhages, or hemorrhagic retinitis, in leukæmic eyes.

In the first group there is a typical affection of the retina which is characteristic of leukæmia. In the second there are either simply hemorrhages as they occur in different organs during leukæmia, or a hemorrhagic retinitis with diffuse dimness of the optic nerve head and retina such as can be seen in diseases of the heart and blood-vessels.

Typical Leukæmic Retinitis, or Papillo-Retinitis.—In this condition the fundus appears of a pale-red, orange-yellow, or even pale-orange-yellow tint in typical cases. Often it is almost lemon-colored, probably because of an accumulation of leucocytes in the chorioidal blood-vessels. This striking color, however, is not seen in all cases, especially in brunettes, whose pigment epithelium is densely pigmented and more or less covers the yellow tint of the chorioidal vessels. The optic disk is but little if at all swollen, and its outlines are partly or completely veiled. There may be a true papillitis, in which the optic disk projects mushroom-like into the vitreous, is opaque, red, and dotted with ecchymoses in stripes. Its outlines also may be invisible, as in papillo-retinitis. Usually the retina is barely opaque. In rarer cases this condition is more markedly so, and in addition it is striped, especially near the nerve head and along the larger blood-vessels. The dimness is rarely so intense as to give it a milky appearance. It may reach the equator, but very seldom approaches the ora serrata. The same is true concerning the swelling of the retina, which is generally but slight.

The retinal hemorrhages, which are variable, are prone to be recurrent. They are often round, but they may be striped, flame-shaped, and even punctiform. Their color is pale red or orange. Most of them are situated in the macular region, where they are generally small. Those lying towards the periphery are usually larger and more numerous. White and more or less prominent plaques with pale-red margins appear in typical cases in about the same area of distribution as the hemorrhages. Large, prominent ones, as Becker[1] has described them in one case, are rare. The retinal blood-vessels are of a pallid hue. The arteries appear pale orange or yellow, while the veins have a pale-rose or an orange-red tint. They are much dilated, are sometimes very tortuous, and often have white borders. (*Vide* Plate XIV.)

Hemorrhagic Leukæmic Retinitis, or Retinal Hemorrhages in Leukæmic Eyes.—In this form, as in retinal hemorrhages from other causes, the ophthalmoscopic picture may vary as regards the number, shape, size, and localization of the blood extravasations. In most cases, however, the conspicuous pale-yellowish color of the fundus is characteristic of the disease. The localization of the hemorrhages in the macular region and towards the ora serrata is a further factor in the differential diagnosis. In retinal

[1] Loco citato.

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hemorrhages the optic disk is normal or slightly pale and its outlines are sharp or indistinct on the nasal side, whereas in hemorrhagic retinitis it is opaque and swollen and its outlines are indistinct. In retinitis the veins are over-filled and tortuous.

The most frequent complications of the condition are considerable dilatation and a pale-yellow color of the chorioidal veins. More rarely chorioidal hemorrhages and hemorrhages with opacities in the vitreous body can be seen. Subconjunctival hemorrhages, subcutaneous hemorrhages in the eyelids, iritis, and irido-cyclitis can all be recognized in proportionally rare cases. Secondary glaucoma and hemorrhagic glaucoma are both found in the grave hemorrhagic types. Lymphomata in the eyelids and exophthalmos due to lymph extravasations or apoplexies in the orbit have also been seen. In one case swelling of the lacrymal gland has been observed.

In leukæmic retinitis the visual disturbances vary greatly. The ophthalmoscope often reveals a typical retinitis while the patient does not complain of visual disturbances. In other cases vision may be diminished. In those without visual disturbances this can be explained by the peripheral localization of the pathological alterations. When there are larger plaques or hemorrhages in the macular region, central or paracentral scotomata may be observed, as Becker[1] has reported in one case. In the hemorrhagic forms vision is always greatly disturbed, and sometimes gradual or sudden blindness develops. Iritic processes and secondary glaucoma may lead to blindness. In all cases in which the optic nerve head is affected it appears swollen, filled with round cells, and is associated with more or less new formation of connective tissue. In some instances, however, this infiltration and new formation of connective tissue may be absent in spite of the swelling, a dilatation of the finer blood-vessels, which are overfilled with leucocytes, being alone seen. At times, especially in purely hemorrhagic cases, this symptom may be absent. In some types the retina, particularly its nerve-fibre layer near the optic disk, becomes infiltrated with round cells. Furthermore, a considerable proliferation of the supporting connective tissue may be found, producing a general swelling of this membrane, particularly of its nerve-fibre layer, in which situation, according to Deutschmann,[2] sometimes a new layer consisting of a connective-tissue net-work containing round cells is formed upon the preformed one. At times, in consequence of a proliferation of the connective tissue, the granular layers appear separated. Hemorrhages are found chiefly in the nerve-fibre layer. Sometimes they are seen in the granular layers or in the intergranular layer. Occasionally they are found on the surface of the retina, whence they project into the vitreous humor. They may penetrate all the layers of the retina to the rods and cones, dislocating or destroying them. The white plaques with red borders seen with the ophthalmoscope consist of accumulations of round cells containing but few red cells in the centre, the latter growing

[1] Loco citato.

[2] Loco citato.

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more numerous towards their margin. They lie mostly in the periphery of the retina, are usually very small and point-shaped, and rarely exceed one or two millimetres in size. They are situated in the nerve-fibre layer or penetrate the whole thickness of the retina, projecting over the inner and sometimes even over the outer surface of this membrane. In rare instances single white plaques which have no red halo, and consisting of sclerotic hypertrophic nerve-fibres, can be found. Foci of fatty granular cells are rare. Saemisch[1] reports a case in which the rods and cones were partly absent. At times the pigment epithelium appears loosened, while rarely it is changed and atrophic.

The larger blood-vessels are dilated and filled with blood-cells, which are chiefly of the white variety. Sometimes the medium-sized vessels contain ectasies in the shape of spindle- or sausage-like dilatations. They are also overfilled, chiefly with white blood-cells. The main alterations found by Deutschmann[2] were in the finer and finest blood-vessels, which were all so overfilled with white blood-cells that they appeared as if they had been artificially injected. He also found enormous dilatations and varicosities in the same vessels and the capillaries. He believes that these enlarged blood-vessels and capillaries filled with leucocytes explain the nature of many of the yellowish-white plaques seen with the ophthalmoscope. The chorioidal vessels are often enormously enlarged and filled with leucocytes. The stroma of the chorioid, too, may contain many leucocytes. Deutschmann sought for micro-organisms in vain. Of course these pathological conditions are not all found in every case of leukæmic retinitis.

In accordance with the primary disease, the course of the retinitis varies.

Leukæmic retinitis is a binocular affection, though both eyes are not attacked equally nor necessarily simultaneously. The frequency of its occurrence in the presence of leukæmia cannot easily be judged. Considering, however, that leukæmia itself is a rare disease, and comparing with this the large series of cases of leukæmic retinitis reported, we may well conclude that retinitis occurs in a rather large proportion of the cases of general leukæmia,—perhaps in from twenty-five to thirty-three per cent., as Leber has stated.[3] On account of the infrequency of the primary disease, the retinitis must be considered a very rare retinal affection. We do not know at what stage of the disease the retinal condition appears. While in many cases the disease has progressed very far before any retinitis develops, instances have been reported by Hirschberg[4] and others in which the visual disturbance was the first recognized symptom of the general disorder. The direct cause of the development of the retinitis must be found in the changed condition of the blood and the blood-vessel walls, which also in other parts of the body leads to hemorrhages and lymphatic infiltration.

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[1] Loco citato.
[2] Loco citato.
[3] Loco citato.
[4] Loco citato.

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As regards the bacillus described by Pawlowsky,[1] it has not been proved that such an organism can produce leukæmia.

In typical cases of leukæmic retinitis the diagnosis is easy. Even in the hemorrhagic form the pale-yellow color of the fundus, the pallor of the blood-vessels and extravasations, as well as their shape and situation, furnish in most cases the most probable differential indications. From this, however, those cases which are like those of hemorrhagic retinitis from other diseases must be excepted, also those instances seen in brunettes in which the yellow color of the fundus is invisible on account of the dark pigment in the epithelium, and in which only an examination of the blood and other changes in the general system can help to secure a proper diagnosis. Such an examination should, of course, never be omitted in any case.

The prognosis of leukæmic retinitis is bad. Thus far, no cure has been reported. Improvements generally do not last, although recently it has been asserted by some observers, especially Mosler, that the prognosis is not necessarily fatal, and that a cure is possible provided the patient receives treatment at a very early stage of the disease. Barrs[2] reports a cure from splenic leukæmia after malaria by means of prolonged exhibition of arsenic and quinine. Should this observation hold good, we might hope also for a possible cure of leukæmic retinitis.

The treatment of leukæmic retinitis is confined to strict dietetic orders, the avoidance of all noxious influences, and the wearing of shaded glasses.

XXVII. RETINITIS OF PERNICIOUS ANÆMIA (RETINITIS ANÆMIÆ PERNICIOSÆ).

This retinal affection has even a briefer history than leukæmic retinitis, since the primary disease, so-called progressive pernicious anæmia, was not recognized as an idiopathic disease and described until 1871 by Biermer.

Of this disease Biermer says that it occurs in females of medium age who generally belong to the poorer classes. The condition is preceded by indigestion or other weakening factors, and is combined with a high degree of emaciation and dropsy. During its course, hemorrhages in the retina, the brain, and the meninges generally occur. Almost without exception the disease terminates in death.

In 1874 Horner,[3] who, on account of the relative frequency of this form of anæmia in Switzerland, had had the rare opportunity of studying thirty cases, stated that he had seen ophthalmoscopic changes in the retina in almost every instance. He found that the picture was often similar to that of leukæmic retinitis, the color of the fundus being changed, the veins being tortuous, and there being numerous hemorrhages.

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[1] Deutsche medicinische Wochenschrift, 1892.
[2] The Lancet, 1891.
[3] Litzenberger ophthalmologischen Gesellschaft für Augenheilkunde, Bd. xiii.

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In 1874 Immermann[1] reported a case which has been ophthalmoscopically examined by Schiess, and which is important because the eye affection was the first symptom of the grave disease. There were hemorrhages along the retinal blood-vessels, with a central yellow discoloration. The papilla was œdematous, swollen, and had indistinct outlines. The œdema reached into the retina. Hæmatogenous pigmentation developed in the hemorrhages and produced a variegated, tiger-skin-like appearance of the fundus.

In 1875 Manz[2] found ophthalmoscopically in one case numerous hemorrhages of a red-brown color, the largest ones being near the papilla and some upon it. Most of them were round and had a distinct whitish centre. The arteries were thin and showed a reflex line. The veins were dark and dilated. The optic disk was pale and had indistinct outlines. The retina appeared dirty gray and opaque. Anatomically there was an aggregation of small round cells surrounded by red blood-corpuscles in the centre of the hemorrhages. The blood-vessel walls were not materially altered. The capillaries, however, showed enlargements or diverticula of varying size and shape, either empty or filled with pale cells or blood-corpuscles.

In one case seen by Quincke there were hemorrhages, white plaques in the circumpapillary zone, and the star-figure around the macula,—conditions characteristic of albuminuric retinitis. In 1879 Litten[3] found accumulations of round cells in the centre of the hemorrhages, œdema of the papilla and the retina, with round-cell infiltration in the retina and blood-vessel walls. In 1880 Uhthoff[4] had occasion to examine anatomically six eyes of four patients in whom, during life, the retinal hemorrhages with white centres had been observed, and found the following factors: hemorrhages in the different layers of the retinæ, varicose hypertrophy of the nerve-fibres, and colloid substances in the intergranular layer.

In my material of observation only five cases of pernicious anæmia with retinal changes have occurred since 1861. Yet even this moderate number of personal observations, together with data from literature, has convinced me that at least in typical cases of pernicious anæmia there is an ophthalmoscopic picture which is more or less characteristic of the disease and which may sometimes at first impress the observer as one of leukæmic retinitis. I do not want to insist that there is any definite symptom that is characteristic,—the hemorrhages with white centres, the blood-vessel alterations, the condition of the papilla and retina, etc.,—all of which may be found in other retinal affections. If, however, the fundus in typical cases is considered,—the peculiar discoloration, the usually round hemorrhages with white centres, the varying color of the hemorrhages from reddish yellow to almost black, the opaque and dirty-looking papilla and retina, the indistinct outlines of the papilla,—and if the anatomical conditions are remembered,

[1] Deutsches Archiv für klinische Medicin.
[2] Centralblatt für die medicinische Wissenschaft.
[3] Berliner medicinische Wochenschrift.
[4] Zeitschrift für klinisch-medicinische Blätter.

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such as round-cell infiltration in the adventitia of the blood-vessels, varices, and diverticula of the overfilled capillaries, which sometimes explain the white spots, the varicose hypertrophic nerve-fibres, colloid bodies in the intergranular layer, etc., it must be evident that we are dealing with a peculiar retinal affection, which has as much right to be considered independently as has leukæmic, albuminuric, or diabetic retinitis. Of course, however, this cannot be maintained for all retinal changes found with pernicious anæmia, perhaps not even for most of them, and in many cases the ophthalmoscopic picture cannot suffice for a correct diagnosis of the primary disease.

The retinal changes due to pernicious anæmia may be conveniently divided into three groups:

(1) *Retinitis of Pernicious Anæmia.*—In this group are placed all cases of retinal changes in pernicious anæmia in which, aside from the hemorrhages, changes occur which must be considered as due to a hyperæmic or an inflammatory condition of the retina. In such rather rare typical cases the optic nerve head is swollen and hazy and its outlines are indistinct. The retina is opaque not only around the posterior pole of the eyeball, but also to the ora serrata. The fundus appears dirty grayish, dim, and livid. Hemorrhages which mostly have whitish centres and are surrounded by distinct red halos are found in the retina, especially around the optic disk, sometimes in it, and in the macula. Usually they are of a round shape, and frequently they show the different changes in color which the blood-pigment ordinarily undergoes. Sometimes single white foci of degeneration independent of the hemorrhages become visible. The arteries are almost empty, and show broad reflex lines. The veins are dark, hyperæmic, and tortuous.

(2) *Retinal Hemorrhages in Pernicious Anæmia.*—The majority of the cases probably belong to this group. In it should be placed all those in which retinal hemorrhages can be observed, but in which no inflammatory signs are visible. The optic nerve head is pale, with fairly distinct or cleanly cut outlines. At times, however, there may be a slight œdema in the retina. The hemorrhages show the same form and arrangement and the same white centres and changes as have been described in the first group. The fundus is generally yellowish in tint, so that from this appearance and its similarity to the condition seen in leukæmic retinitis a probable conclusion as to the primary disease can in most cases be arrived at. There are, however, cases in which neither the color of the fundus nor the arrangement of the hemorrhages shows anything that is characteristic, so that it is impossible to distinguish such a retinal hemorrhage by the ophthalmoscope alone from that of other origin.

(3) *Albuminuric Retinitis in Pernicious Anæmia.*—In this group belong the well-known case of Quincke, one of Galezowski, and several others, in which the ophthalmoscope revealed a condition almost identical with that found in typical cases of albuminuric retinitis. Aside from hemorrhages,

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this type shows a swollen and dim optic disk and retina, the outlines of the nerve head being indistinct. In the circumpapillary zone white plaques varying in size and number can be found. There are star-like figures around the macula. The veins are broad and tortuous, but there are no pathological changes in the periphery of the retina. Concerning this rare example, two possibilities must be thought of: either that there is an albuminuric retinitis which has developed in a subject suffering from pernicious anæmia and which is independent of the latter disease, or that there is a noxious agency in pernicious anæmia which alone can, in exceptional cases, produce such retinal changes as are generally ascribed to albuminuric retinitis. If this latter supposition be granted, then these cases fall in line with those rare types in which during other diseases, as cerebral tumors (Schmidt-Wegner), lead intoxication (Lehmann and others), the typical picture of albuminuric retinitis that is independent of any nephritic process and albuminuria is observed.

Visual disturbances vary greatly, bearing no apparent relation to the amount or degree of the ophthalmoscopic change. Central scotomata have been seen.

Owing to the early occurrence of death from pernicious anæmia, it is not astonishing that, in comparison with the rarity of the disease, anatomical examinations are not very scarce. The most important ones are those of Uhthoff and Manz.[1] The optic nerve and the retina are sometimes swollen and opaque. Usually œdema, with rarely round-cell infiltration, is found. Retinal hemorrhages are seen without exception, being generally situated near the disk and the large blood-vessels. In most cases the size of the hemorrhages is not considerable, the largest being about a quarter the area of the disk. Their shape is round, and their tint is dirty yellowish or brown-red. Most of them have a white centre, which at the first appearance gives the fundus somewhat the picture of a leukæmic retinitis. According to Litten,[2] these white centres consist of accumulations of round cells, due to varicose enlargements of the capillaries. Uhthoff,[3] however, has not, from an examination of his own cases, been able to agree with these statements. Besides the hemorrhages, there are plaques of varicose, hypertrophic nerve-fibres (Uhthoff) lying in the nerve-fibre layer and projecting sometimes into the vitreous humor. At times the hemorrhages are of considerable size and contain shining finely granular bodies, which are shaped like a sphere, a spindle, or a retort. Sometimes one fibre exhibits several varicosities; others have one or two branches, and appear like unipolar or bipolar ganglionic cells. These foci are generally isolated from the hemorrhages. In some places, however, they appear to be surrounded by a hemorrhagic halo, or red blood-corpuscles may be found between the nerve-fibres. Uhthoff has explained the white centres of the hemorrhages, as seen with the ophthalmoscope, as a varicose hypertrophy of the nerve-fibres, proving

[1] Loco citato. [2] Loco citato. [3] Loco citato.

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the inflammatory nature of these cases. He has also found peculiarly glistening colloid or finely granular bodies in the intergranular layer, as they have been determined to exist in albuminuric retinitis. These appear in the shape of irregular, variously sized lumps, or as conglomerations of round bodies, etc. This condition, too, convinces Uhthoff of the inflammatory character of the disease. The adventitia of the blood-vessels shows no particular changes, yet blood-corpuscles have been found in it. (Natanson.[1]) The capillaries sometimes present fatty degeneration. Blood and serum are not infrequently found between the retina and the chorioid. (Sargent.[2])

The course of retinitis of pernicious anæmia is governed by that of the primary disease, which almost without exception leads to death in a few months or in a year or more. It must not be forgotten, however, that the development of the primary disease is very slow, so that its beginning is not even known to the patient, who comes to the physician later in the progress of the disorder. If, as in rare cases, an improvement takes place, the visual disturbance remains. Later the betterment disappears and the grave disease returns, gradually leading to the highest degree of emaciation and to death.

In general the prognosis is bad, since the primary disease soon leads to death and cures are of the greatest rarity. Moreover, it seems that the gravity of the retinal changes, particularly as regards their number and their extent, has a bad prognostic value for the primary disease, and it may be possible from them to decide the malignity of the affection.

The treatment consists in that which is directed towards the primary disease.

XXVIII. DIABETIC RETINITIS (RETINITIS DIABETICA—GLYCOSURIA MELITURICA).

Diabetic retinitis is a much rarer affection than albuminuric retinitis, and was much later recognized as the real cause of the long-known visual troubles accompanying the general disease. There are even to-day a number of prominent ophthalmologists who do not consider it proper to assign to diabetic retinitis a separate place among the diseases of the retina, and either ignore it or deal with it in a few brief remarks, as, for instance, such investigators as Schweigger, Schmidt-Rimpler, and Jacobson. In my very rich material there have been but a few undoubted cases, about nine in number. Most of these were seen but cursorily, because they came to the dispensary service and, as a rule, did not return. Almost all of them, however, agreed with the description given by Hirschberg, thus convincing me that diabetes produces a special form of disease of the retina, which in most cases can be differentiated from other forms of retinitis.

From a study of the published cases and my individual observations, Hirschberg's division, especially that of his two chief forms, has been found to be the best: (1) central punctate diabetic retinitis (Hirsch-

[1] Loco citato. [2] Loco citato.

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berg), which I consider to be the typical form of the disease; (2) hemorrhagic diabetic retinitis (Hirschberg); (3) diabetic albuminuric retinitis, which is a mixed type situated between the first two forms, and which has been described by Dahrenstädt, reported by others, and personally seen; (4) albuminuric retinitis in eyes of diabetics. Under this head the writer includes numerous cases in which in diabetics considerable albumen is found besides sugar, and the ophthalmoscopic picture is very similar to that of albuminuric retinitis and can seldom be differentiated from its typical forms. (5) Atypical diabetic retinitis. Under this head I include the cases which Hirschberg places in his third group, and among which he relates three instances of pigmentation of the retina with contraction of the visual field and night-blindness. Of these symptoms he remarks, however, that their causal connection with the primary disease is not clear.

In addition there is a form of diabetic retinitis which I have seen and of which I have found but one case in literature. In this form larger and smaller white plaques were observed all over the retina and as far forward as the ora serrata; also the rarely mentioned cases of chorio-retinitis and diabetic neuro-retinitis.

(1) *Central Punctate Diabetic Retinitis* (*Hirschberg*), *or Typical Diabetic Retinitis.*—In this type the optic nerve head is neither swollen nor clouded. The retina is not diffusely opaque, nor is it swollen. At the posterior pole of the fundus, especially in the macular region, are found numerous ivory-white points, spots, and stripes which surround the macula in an irregular manner without forming the regular radiating or star figure which is presented in albuminuric retinitis. These spots are round or oval or are irregular in outline; sometimes they are linear or semilunar in shape. Their margins are often serrated. Smaller aggregations of these spots are seen between the macula and the optic nerve head up to the superior and inferior temporal vessels of the retina. Beyond these vessels they may appear as single spots, but they are much rarer on the nasal side of the disk. Some of them cover small blood-vessels, which shows that they lie in the nerve-fibre layer. Rarely a blood-point is seen in one of them. Larger white spots are of very infrequent occurrence. They appear to remain unchanged for years; their number may increase, yet they never coalesce and form larger plaques or group themselves around the macula in radii. Everywhere between these white areas fine blood-points, stripes, or little spots (and rarely somewhat larger hemorrhages) may be seen. These blood-spots usually reach farther to the periphery than the white spots; they also extend farther on the nasal side of the disk and beyond the temporal vessels. The periphery of the fundus appears normal; there are no pigment alterations. The vitreous does not contain any opacities or hemorrhages.

(2) *Hemorrhagic Diabetic Retinitis* (*Hirschberg*).—In this form extravasations of blood are almost solely found, but no groups of white spots are visible. As in the preceding type, the disk is sharply defined. The retina and the blood-vessels, particularly the large ones, appear normal.

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Four different types of hemorrhagic diabetic retinitis may be distinguished:

(*a*) Sometimes fine punctiform hemorrhages will be accidentally found when for some reason, as for refractive work, an ophthalmoscopic examination is made, thus inducing an examination of the urine, when an unsuspected diabetes will be discovered.

(*b*) Larger hemorrhages, several millimetres in diameter, which sometimes lie farther towards the periphery of the retina, and may enter the vitreous humor and there produce a localized bluish dimness.

(*c*) Sudden large hemorrhages in cases in which there is even very little sugar in the urine. These, fortunately, are but rarely observed.

(*d*) Hemorrhagic glaucoma, permitting no hope of recovery.

Probably, however, it would answer the purpose better to term this type of affection retinal hemorrhages in diabetes instead of retinitis, since in these cases no symptoms nor processes of inflammation are observed. Furthermore, it must be understood that there are mixed forms of the former and this type of disturbance in which numerous hemorrhages, together with white spots, are observed.

(3) *Diabetic Albuminuric Retinitis.*—This form presents a mixture of typical diabetic retinitis and albuminuric retinitis. Just as in analyzing the urine albumen and sugar may be found, so there are cases in which an ophthalmoscopic examination reveals the signs of albuminuric retinitis superadded to the typical picture of diabetic retinitis. In these cases, besides an opaque and slightly swollen optic nerve head and retina, and alterations of the blood-vessels that are characteristic of albuminuric retinitis, there are groups of white spots which are peculiar to diabetic retinitis.

Dahrenstädt has reported such a case[1] and has given an illustration of it.

(4) *Albuminuric Retinitis in Eyes of Diabetics.*—It is well known that forms of retinitis can be seen in diabetic subjects which agree in all essential points with the picture of albuminuric retinitis. Since it is known that in many, perhaps in most, protracted cases of diabetes an affection of the kidneys with albumen may develop, and since retinitis is very rarely seen in diabetes, it may with great probability be assumed that in these cases the secondary nephritic process which complicates the diabetes produces its own characteristic form of retinitis,—namely, an albuminuric retinitis. In these cases, therefore, there is a purely albuminuric retinitis which is independent of the diabetic process, except that it appears in a diabetic subject. Therefore I think the term albuminuric retinitis in diabetic eyes or diabetic subjects will appear to be the proper one.

(5) *Atypical Diabetic Retinitis.*—Under this head must be included the cases which Hirschberg places in his third group. These were three cases of pigmentation of the retina with contraction of the visual field and night-

[1] Centralblatt für Augenheilkunde, 1892, S. 132.

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blindness seen in diabetic subjects, concerning which Hirschberg himself states that the causal relation between the eye-affections and the diabetes was not well proved. It seems to me that they were cases of pigmentary retinitis in which diabetes accidentally developed subsequently.

To these cases I may add one which I observed in a diabetic subject seen in 1865. The patient was an elderly official in a factory, who called on me because he was gradually losing the faculty of reading and writing and wanted proper glasses. No lens gave him any improvement. Ophthalmoscopically I found in the fundus of each eye large white plaques, some of which were round, others polymorphous. These massings were distributed irregularly from the optic disk to the equator of the eye, some even reaching almost to the ora serrata. In addition, an irregular aggregation of small white spots was found in both macular regions. On the temporal sides of the retinæ there were a number of blood-points and stripes, and in the left macular region there was a somewhat larger hemorrhage. The optic nerve head was well defined. The retina, too, was neither dim nor swollen, and the blood-vessels were normal. Examination of the urine failed to show albumen, though there was three per cent. of sugar. The patient did not suspect the serious nature of his ailment. By my advice he underwent a Carlsbad cure, and the amount of sugar was considerably lessened, to recur soon afterwards, and then to disappear. The ophthalmoscopic picture and the visual disturbance remained unchanged. The patient had to resign his office, and died two and a half years afterwards. From the ophthalmoscopic appearances detailed above, and from the fact that up to his death not a trace of albumen could be found in his urine, I considered this to be a case of uncomplicated diabetic retinitis.

The ophthalmoscopic appearances in this case are reproduced on Plate XV., from my former sketches, by my son. On Plate XVI., for comparison, I have had reproduced an example of typical diabetic retinitis from Hirschberg's sketch,[1] since I have not seen a more typical one myself.

For a definite description of the symptom-complex of this group the number of cases observed is as yet much too small, and in the future, when further observations may be reported, this type will perhaps have to be divided into several more sharply defined special conditions.

The cases in which the subjective visual disturbance is altogether out of proportion with the objective ophthalmoscopic appearance must be considered as being complicated with amblyopia. As we know that amblyopia without any ophthalmoscopic signs and without the development of a retinitis is not a rare occurrence in diabetes, I think this assumption will appear correct. This belief also gives an explanation of those rare cases of contraction of the visual field found with diabetic retinitis and which are not explainable by the ophthalmoscopic appearances alone.

Amblyopias of different degree, with or without contraction of the visual

[1] Centralblatt für Augenheilkunde, 1891.

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field or central scotoma, may be found. Concerning the amblyopias with central scotoma, which latter cannot usually be explained by the ophthalmoscopic appearances alone, it seems probable that, as Mauthner has recently asserted,[1] in the majority of such cases the central scotoma is not produced by the diabetic process, but is the result of the abuse of tobacco and alcohol. There is, however, an indirect connection between these cases and the primary disease, since the latter generally renders the organism less resistant to these poisons, and thus a central scotoma becomes proportionally more frequent in diabetics than in healthy subjects, even when there is no disease of the retina.

Atrophy of the optic nerve head, with progressive amblyopia and concentric or irregular contraction of the visual field and disturbances in color-perception, is at times seen. The development of visual disturbances, especially of amblyopia, is extremely frequent in diabetic subjects, and leads in many instances to the discovery of the serious general disease while the patient still feels well. Therefore an examination of the urine should not be omitted in any case of amblyopia, since the earliest recognition of the disease is of paramount importance for the patient's welfare.

Opacities and hemorrhages of the vitreous body are frequently found in association with diabetic retinitis. Chorioiditis and optic neuritis, when accompanying diabetic retinitis, produce the ophthalmoscopic appearances of chorio-retinitis and diabetic neuro-retinitis, as described under the head of atypical diabetic retinitis.

Hemorrhagic glaucoma in the fulminant form may appear primarily in diabetics, or may develop later after a prolonged course of a diabetic retinitis, especially the hemorrhagic form.

Diabetic patients frequently complain early of a glimmer before their eyes. Soon a disturbance of central vision makes its appearance, so that they can read only with difficulty and later not at all, their peripheral vision remaining undisturbed. In some types of protracted cases the visual acuity may fall to one-half or one-third or even one-tenth of normal with a good visual field, even where no complication with amblyopia or neuritis or atrophy of the optic nerve is present. Sudden diminution of vision is generally produced by hemorrhages. Photopsiæ, chromopsiæ, and metamorphopsiæ are not very frequently complained of. This is true also of disturbances of color-perception with contraction of the visual field, which are usually accounted for by one of the complications mentioned above. The contraction of the field of vision may be concentric or concern one sector only. Central scotoma is not infrequently observed, but, as Mauthner says, it is generally produced by the abuse of tobacco and alcohol.

Diabetic retinitis is a very chronic affection, which may exist unaltered for years. It is rarely improved, and usually becomes worse from time to

[1] Wiener medicinisch-klinische Rundschau, 1893.

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time. Total blindness is probably never produced by typical diabetic retinitis, yet it is quite frequent in the hemorrhagic form or when there is hemorrhagic glaucoma. As a rule, diabetic retinitis develops when diabetes has been in existence for a long period. It is always binocular, although it is not necessary that both eyes should be attacked simultaneously. It is difficult to form a decisive opinion as regards the frequency of the development of retinitis in diabetics in general, because the data concerning the number of cases of diabetes treated and of retinitis observed among them by clinicians are as yet too meagre, and ophthalmologists see only some of those diabetic subjects in whom vision is disturbed. Seegen[1] found but two cases of retinitis in one hundred and forty diabetic patients.

Even from the ophthalmoscopic picture alone the diagnosis of the typical form of diabetic retinitis is not difficult. The most important points in distinguishing it from albuminuric retinitis are: the unaltered condition of the papilla, the lack of any swelling and dimness of the retina in the posterior polar region, especially around the optic nerve head, and the absence of alterations of the blood-vessels. It is not likely that diabetic retinitis can be confounded with specific chorio-retinitis, which, although appearing at times as a central affection with small foci, is always accompanied by changes in the pigment epithelium. Retinitis punctata albescens (Mooren) may sometimes produce a similar ophthalmoscopic appearance, yet the general picture is different in the facts that the spots lie behind the blood-vessels and that there is no sugar in the urine.

From the ophthalmoscopic picture alone it is often impossible to make a differential diagnosis of hemorrhagic diabetic retinitis in all of its four groups, as an ophthalmoscopic examination may be rendered impossible on account of numerous hemorrhages in the vitreous humor in the hemorrhagic glaucoma type of the affection, or the ophthalmoscopic picture may agree with that of hemorrhages in the retina from other causes. In these cases the presence of sugar in the urine alone is the deciding symptom in the diagnosis.

The development of a diabetic retinitis is always a bad sign in regard to the primary disease. On the other hand, the appearance of amblyopiæ without ophthalmoscopic signs, as sometimes occurs in the beginning of the primary disease, must be looked upon as a benefit to the patient, since through such signs the attention of the physician is directed to the underlying serious disease, and by means of proper therapeutic measures its course may be modified.

Diabetic retinitis, however, mostly appears after the general disease has been in existence for a long period of time, and when the condition is recognized the alterations in the fundus of the eye are of such a nature that little can be hoped for from therapeutic measures. The prognosis of hemorrhagic diabetic retinitis is much worse than that of the typical form,

[1] Diabetes Mellitus, Leipsic, 1870.

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since the former is usually due to atheroma of the blood-vessels. In hemorrhagic diabetic glaucoma the prognosis is hopeless.

The treatment of diabetic retinitis is covered by the treatment of the diabetes. In the first place an appropriate diet which aims at an exclusion of sugar and starches—the so-called Cantani's diet—is necessary. This permits the ingestion of beef (roast), game, fowl, ham, butter, cheese, green vegetables, gluten bread, almond bread; while for drinks, mineral waters, coffee, tea without sugar, and liquors and wines which are not sweet are indicated. It excludes sugar, the starches, chocolate, sweet wines and liquors. Further most efficient remedies are the water-cures, particularly those like Carlsbad and Vichy. Gymnastics and massage are also useful. The most highly recommended medicaments are pills containing carbolic acid, lithium, or arsenic. Ergotine subcutaneously employed is valuable, while strychnine, iodide of potassium, salicylate of sodium, antipyrin, salol, and thymol have all been recommended. Jambol, made from the root of the East Indian *Syzygium Jambolana*, has not yet been tried sufficiently. Oxygenated water has not been altogether unsuccessful. In recent years saccharin has been recommended frequently in order to allow small quantities of the starches to be eaten with impunity, but it is usually not well borne for any length of time.

XXIX. ALBUMINURIC RETINITIS (RETINITIS ALBUMINURICA).

It is a well-known fact, as has been mentioned by Leber[1] in his historical introduction, that the occurrence of visual disturbances accompanying conditions producing dropsy, such as scarlatina, pregnancy, and the puerperium, had been observed a long time before Bright, who in 1827 ascertained the connection between these dropsies and a disease of the kidneys, and reported a series of cases in which he and Barlow had observed similar visual disturbances developing in the form of albuminuric nephritis called after him.

In typical cases the ophthalmoscopic appearance of albuminuric retinitis is characteristic. These, however, are among the minority of those observed; and even in them we are not able to judge the nature of the retinal affection with absolute correctness, since instances have been reported in which, in spite of a picture which was formerly considered to be pathognomonic of the condition,—viz., a wall- or star-like arrangement of white spots around the macula,—no albuminuric retinitis was present. Especially is this so with intra-cranial tumors, where an ophthalmoscopic picture, as was shown by a highly interesting case of Schmidt and Wegner,[2] may be observed. In this instance, in which an albuminuric retinitis was believed to be present for a protracted period, the post-mortem examination revealed an intra-cranial gliosarcoma. Such pictures have been found also in diabetes.

[1] Graefe und Saemisch, Handbuch der gesammten Augenheilkunde, Bd. v.
[2] Archiv für Ophthalmologie, Bd. xv., 3, S. 253.

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The ophthalmoscopic picture which is generally found in the different forms of albuminuric retinitis is the following. The optic nerve head appears opaque, reddish, and swollen, its limits not being clear and in some cases not even being visible. The posterior portion of the retina appears as a light pale-gray membrane with opaque stripes. A peripapillary zone which may surround the disk with a diameter of from four to six times its own size shows some extravasations of blood, which are mostly flame-like or in stripes and vary in size and number. In the same zone white or yellowish spots, varying in number and size, sometimes even surrounding the optic nerve head, may be found. At times, in this zone and a little outside of it, small, silvery, shining points can be seen. The macula is often red and is surrounded by a wall of white foci of degeneration or by a characteristic star- or halo-like arrangement of spots. The arteries are usually thin, and are often outlined by whitish stripes. The veins are broad, of a dark-red color, and more or less tortuous. These are recognizable or not according as their tortuous course brings them nearer to the surface or carries them deeper into the tissue. In uncommon cases various changes in the chorioid, especially in the pigment-layer, are found. These are followed by changes in the vitreous humor, such as opacities and hemorrhages. Further strange alterations in the optic nerve head can be seen, followed finally by retinal detachment.

The following forms can be differentiated: 1, typical albuminuric retinitis; 2, degenerative albuminuric retinitis; 3, hemorrhagic albuminuric retinitis; 4, albuminuric chorio-retinitis; 5, albuminuric neuro-retinitis; 6, albuminuric papillitis; and, 7, saturnine retinitis. Of course these different forms may be seen to merge into one another, and it may sometimes be very difficult to place a case in any special grouping.

1. *Typical Albuminuric Retinitis.*—In this group might be placed above all others the well-known case which Liebreich has described and drawn in the *Archiv für Ophthalmologie*,[1] and which has found its way into most text-books as a prototype of albuminuric retinitis, although it has been stated by Magnus,[2] Mauthner,[3] Schweigger,[4] and Szokalski[5] that this very case of Liebreich's cannot be considered as a prototype for all or even for the majority of cases of albuminuric retinitis, it being one of the rarer forms.

The characteristic features of this group are a moderate inflammatory change of the optic nerve head, an average number of hemorrhages, a collection of white foci of degeneration, which later coalesce into a more or less continuous zone around the disk, and, finally, characteristic alterations situated in the neighborhood of the macula.

We may, as Leber[6] did, distinguish between three stages in this group, —namely, the hyperæmic or hemorrhagic, the degenerative, and the atrophic.

In the first the optic nerve head is uniformly red and opaque and its

[1] Archiv für Ophthalmologie, Bd. xv., 2, S. 265.
[2] Loco citato. [3] Text-Book. [4] Ibidem.
[5] Wyklad chorob Warzawa, 1869. [6] Loco citato.

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borders are indistinct, it not being markedly swollen. The retina, especially where it surrounds the disk, is opaque and of a grayish-red color. Small hemorrhages, at first scarce and generally flame-shaped, appear in the nerve-fibre layer, on the nerve head itself and in its neighborhood, especially along the blood-vessels, and frequently also in the macular region. The number of these hemorrhages gradually increases, while sometimes larger, irregularly shaped ones that lie in the deeper layers make their appearance. Of the white foci of degeneration, only the prodromes in the shape of infrequent minute white spots or points, first appearing around the macula, are seen at this stage. The blood-vessels show the signs of an advanced venous hyperæmia. The arteries are either normal or somewhat narrowed. At times they have white borders. The veins are considerably enlarged, dark red in tint, and very tortuous. In places they are covered by opaque tissue, and therefore appear interrupted in their course. The equatorial and peripheral parts of the retina towards the ora serrata are more or less normal. As complications we sometimes observe opacities or hemorrhages in the vitreous body. In addition, a circumscribed peripheral detachment of the retina may develop, as Liebreich has already mentioned and depicted in his case above referred to, and as has been recorded in literature (Schlesinger, Kraskowsky, Lutz, etc.).

In the second stage the symptoms of hyperæmia and swelling of the retina diminish, and the reddish-gray dimness in the neighborhood of the optic nerve head gives place to a more or less bluish-gray, sometimes striped, opacity. Part of the hemorrhages become almost invisible, while some new ones occur which in this stage, too, appear at first along the course of the blood-vessels, though sometimes in the centre of some white plaques (when they have a white border). At first small, then larger and larger white spots or foci of degeneration make their appearance, which in the typical cases lie in a circumpapillary zone. They vary in size from a pin-head to the extent of the disk itself, though sometimes they are much larger. Their shape differs greatly. Their borders are often sharply defined and are frequently indistinct. Sometimes the small ones develop apparently quite suddenly; the larger ones, as Magnus[1] states, seem to contract to white masses from the diffuse bluish-gray opacity of the retina. At first they can barely be distinguished from their surroundings. Later they increase in intensity and become shining in appearance, and finally they spring forth from the darker background as bright white plaques. It is rare to find any plaques in the region of the equator and outside of the circumpapillary zone. Between these plaques, and situated outwardly from them, little white points may be observed. At an early period minute white points or stripes group themselves in the shape of a star, a rosette, or a halo around the macula, growing gradually in numbers and size, and sometimes coalescing to a continuous ring around the macula.

[1] Loco citato.

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Later the white plaques in the zone of degeneration around the optic nerve head become more numerous and larger, several of them joining together to form still larger ones, finally forming a continuous white wall, which at times remains open on the side towards the disk. This second stage may last for a prolonged period, sometimes as long as life.

In the third stage the gray dimness of the fundus gradually diminishes. The white plaques change slowly to dirty-white or grayish-white ones, and their margins become less sharp and indistinct. Finally they may almost all disappear, so that but a few dirty-white spots remain, and something like a thin veil appears over the fundus, which has regained its red color. The star-figure around the macula probably lasts the longest. The optic nerve head becomes of a dirty-white, or whitish-gray, or yellowish-white tint (retinal atrophy). The blood-vessels grow very narrow, especially the arteries, which are sometimes bordered by white lines, or, no longer carrying blood, are represented by whitish dendritic filiform bands, so that at times they seem to be wanting altogether either in parts or in the whole of the retina, the fundus appearing to be without blood-vessels. In this stage degenerative alterations frequently take place in the pigment-layer.

It is probably in rare instances only that such a termination of an albuminuric retinitis is observed, since in the vast majority of cases the general disease destroys the patient before any such changes can take place in the retina.

Notwithstanding my enormous material for observation, I have seldom seen this proportionately rarer form in all its details. I gave it the name of typical, not because it represents the type of all or of the majority of the cases, but because for decades, since its first description by Liebreich, it has been looked upon by most ophthalmologists as the prototype of albuminuric retinitis, and has been and still is described as such in most text-books. In this way it has, so to speak, gained citizenship and the right of a home in ophthalmology as a prototype.

2. *Degenerative Albuminuric Retinitis.*—This group has been given its proper place by Magnus,[1] whose description we follow. It probably comprises by far the greater number of cases of albuminuric retinitis which come under observation. The characteristic features of this group are the prevalence of white foci of degeneration, the absence in most cases of the continuous white ring in the peripapillary zone, the frequent lack or incomplete irregular development of the star-figure around the macula, and the low degree of swelling of the optic nerve head.

In the first stage the disk is red and swollen and its margin is indistinct. The retina appears gray, opaque, and swollen, especially around the nerve head. The blood-vessels, particularly the veins, are very tortuous, and this tortuosity is either confined to the swollen and opaque circumpapillary zone or extends over the fundus. The blood-vessels are more or less

[1] Loco citato.

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opaque, as if veiled, in this zone of the retina. Alongside of the blood-vessels, sometimes on them or between them, appear first small and afterwards large, striped, fusiform, or flame-shaped extravasations of blood. In the macular region sometimes irregular extravasations can be found.

In the second stage the white plaques which have been described in the preceding group begin to form in the swollen and opaque retina. The form, size, and margins of these plaques vary greatly. Sometimes they cover some of the blood-vessels, while at times small silver-white, glistening points or spots can be seen.

The white wall around the papilla is exceptionally seen. In most instances it is absent, and the small white spots coalesce in places to form large irregular polymorphous plaques. The regular star-figure of white points or stripes around the macula is frequently wanting.

When the star-figure is present, or when a continuous ring has been formed, the macula appears at first darker red and later grayish white with an admixture of green, and thus strongly contrasts with the shining white ring. This contrast is lost later, and the corresponding portion of the retina appears uniformly white.

The third stage, which is seldom seen, is similar to that detailed in the former group.

3. *Hemorrhagic Albuminuric Retinitis.*—This is a comparatively rare form of the disease. It is characterized by a great tendency to extravasation of blood, while the optic nerve head and retina are otherwise normal. Rarely small white plaques make their appearance.

Such cases have been described by Holmes,[1] Niemeyer,[2] Wegner,[3] Hirschberg,[4] and Magnus.[5] I have seen three examples, two of which differed from those seen by other observers in that the optic disk was quite red and its borders were indistinct.

All the cases were found in aged people, most of whom suffered from arterio-sclerosis, thus giving a bad prognosis to the affection, as, aside from the hemorrhages in the eye, apoplexy in the brain must be expected. In every case the nerve head either is of normal color and sharply defined or is moderately red with somewhat indistinct contours. It is never swollen. The retina is neither opaque nor swollen. Ophthalmoscopically the blood-vessels show no pathological alterations. On the other hand, hemorrhages, especially those of the commoner varieties, which are localized in the nerve-fibre layer,—linear, in stripes, fusiform, and flame-shaped,—are numerous. Later larger hemorrhages of various shapes can be seen in the deeper layers. These hemorrhages are generally found alongside the blood-vessels, and, while some disappear, new ones constantly occur. Finally they may be absorbed, or white spots or hæmatogenous pigment remain in their stead if

[1] British Medical Journal, 1857. [2] Lehrbuch, 1865.
[3] Archiv für pathologische Anatomie und Physiologie und für klinische Medicin, Bd. xii.
[4] Berliner klinische Wochenschrift, 1870. [5] Loco citato.

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the patient survive sufficiently long. At times a few small yellowish spots may be present.

4. *Albuminuric Chorio-Retinitis.*—This affection is now and then observed, and in it the chorioidal alterations are generally combined with the degenerative form of albuminuric retinitis.

The characteristic feature is the appearance of more or less numerous grayish-black or deep-black pigment spots and lumps, which ordinarily form a border around the white plaques in the zone of degeneration. Although alterations in the chorioid have been found in most cases of albuminuric retinitis which have been examined anatomically, they can be demonstrated ophthalmoscopically in proportionately few instances. In such cases pigment-spots similar to those seen in disseminated or, rather, areolar chorioiditis will be found added to the ophthalmoscopic picture of degenerative albuminuric retinitis. If the accumulations of pigment are very small and enter the retina, a picture may result which is quite similar to that observed in pigmentary retinitis.

This affection is sometimes complicated by the appearance of crystals of cholesterin in the vitreous humor.

There is also an independent form of albuminuric chorioiditis which Magnus[1] has described, and which cannot be explained at length among the diseases of the retina, as it belongs to the chapter on chorioidal diseases.

5. *Albuminuric Neuro-Retinitis.*—This not particularly infrequent affection is characterized by a moderate inflammation of the optic nerve head—*i.e.*, neuritis—which is added to the albuminuric retinitis. Since this may happen conjointly either with a degenerative albuminuric retinitis or with a hemorrhagic albuminuric retinitis, we can differentiate between these two forms. Such cases have been observed by Schweigger,[2] Wegner,[3] Agnew, Schlesinger,[4] Oliver,[5] and Bull.[6]

It seems that in these cases the neuritis is a secondary one and due to the retinal affection. The optic nerve head shows the signs of a moderate neuritis. The disk is red, opaque, and swollen, and at times is sprinkled with ecchymoses. To this is joined the picture of either a hemorrhagic or a degenerative albuminuric retinitis.

6. *Albuminuric Neuritis or Papillitis.*—This affection is very rare, and gives the well-known picture of a choked disk with an otherwise almost intact retina.

Such cases have been described by Heymann,[7] Liebreich,[8] de Wecker,[9] Schmidt,[10] and Magnus.[11]

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[1] Loco citato.
[2] Handbuch.
[3] Archiv für Augenheilkunde, Bd. xv., 3, S. 253.
[4] Beiträge, Berlin, 1884.
[5] British Medical Journal, 1885.
[6] Transactions of the American Ophthalmological Society, 1893.
[7] Archiv für Augenheilkunde, Bd. iv., 2, S. 131.
[8] Atlas.
[9] Traité complet d'Ophtalmologie, Paris, 1886.
[10] Berliner klinische Wochenschrift, 1870.
[11] Loco citato.

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In these cases the optic nerve head, which is of a grayish-red color, appears extremely swollen, protruding from the fundus like a toadstool. It is covered with whitish spots and extravasations, and the blood-vessels form steep arches. The veins are very broad and dark in tint. In different places the blood-vessels may disappear into the opaque nerve head. The retina shows signs of hyperæmia, a higher degree of fulness, and a vascular tortuosity, especially of the veins. Rarely a hemorrhage or a few small yellowish-white spots can be found.

In the later stages papillitic atrophy develops, the optic nerve head assuming a dirty color and appearing larger than its normal boundaries.

It is likely that the products of the primary disease, as affections of the meninges or of the sheath of the optic nerve, give rise to this rare form. These cases also teach us that papillitis should always induce an examination of the urine for albumen.

7. *Saturnine Retinitis.*—Concerning this affection, of which it is well known that it generally presents a picture similar to that of albuminuric retinitis, and that it is often in no way to be distinguished from this by the ophthalmoscope, it is necessary to state that this form of retinal affection, following Remak[1] and Jacobson,[2] has been declared to be due to nephritis with albuminuria complicating lead intoxication by such eminent ophthalmologists as von Graefe, Leber, Michel, Schmidt-Rimpler, and Meyer. Daujoy, Lancereaux,[3] Deprey,[4] Stephan,[5] Pedell,[6] and Oliver[7] have also described such cases.

More recently Förster has stated that retinitis from lead intoxication can occur independently of any kidney affection, and particularly albuminuria. He also says that it may happen in cases of lead intoxication in which such complications are never seen. Rosenstein[8] is of the same opinion, believing that lead acts directly upon nerve-substance. A case observed by Lehmann[9] confirms this explanation. Knies,[10] too, is of this opinion.

We should, therefore, not consider saturnine retinitis when treating in general of albuminuric retinitis; yet it must not be forgotten that during lead intoxication, even when there is no albuminuria, ophthalmoscopic symptoms may be observed that are similar to those found in albuminuric retinitis, just as has been observed by Schmidt and Wegner[11] to happen in cerebral affections.

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[1] Berliner klinische Wochenschrift.

[2] Bericht der Sehorgane bei allgemeinen Krankheiten, 1885.

[3] Schmidt's Jahrbücher, 1864.

[4] Gazette des Hôpitaux.

[5] Bericht.

[6] Loco citato.

[7] Loco citato.

[8] Archiv für pathologische Anatomie und Physiologie und für klinische Medicin, Bd. xxxix.

[9] Loco citato.

[10] Die Beziehungen des Sehorgans und seiner Erkrankungen, 1893.

[11] Loco citato.

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Aside from the complications in the retina itself, above mentioned, amotio retinæ, which has been seen by Andersen,[1] may be found; also embolism of the central retinal artery, as has been recognized twice by Völckers;[2] opacities and hemorrhages of various kinds, and more or less serious changes in the chorioid and optic nerve head. Plastic iritis is an extremely rare complication. The two cases in which I saw this condition suffered from severe albuminuric retinitis, but I did not have the opportunity to observe them for any length of time. In the first case the patient had diffuse opacities in the most posterior parts of the vitreous humor. He had had uræmic attacks before consulting me. His urine contained much albumen. I found a plastic iritis which had developed without the least irritation or ciliary neuralgia. In two places there were posterior synechiæ, although the iris was not discolored, just as is sometimes seen with subretinal cysticercus or in the beginning of intra-ocular tumors. Unfortunately, I saw the patient but twice. I do not doubt that I had here to deal with an insidious irido-chorioidal process, and that had the patient lived, progressive atrophy of the eyeball would have resulted. Such cases must not be confounded with those in which an albuminuric retinitis supervenes in an eye with old synechiæ.

Further, like Talko and Samelsohn, I have seen repeated subconjunctival hemorrhages, and once I found a case with a hemorrhage into Tenon's capsule and the orbit, as has been described by Wharton Jones.[3] It is difficult to decide whether senile cataract, which I have observed several times with albuminuric retinitis, is in any manner due to the kidney-lesion or to the eye-affection. Other complications are uræmic amblyopia and amaurosis. Severe attacks of epistaxis and cerebral apoplexy may be found with albuminuric retinitis. Hemorrhagic glaucoma has been observed by de Wecker and Mooren.[4]

The visual disturbance due to albuminuric retinitis differs much in degree, it not being in proportion to the ophthalmoscopic appearance nor to the kidney-disease. It is not always the same in each eye. Improvement and restitution of vision are possible if the primary disease be improved or cured: especially is this true of albuminuric retinitis found during pregnancy, childbirth after scarlatina, and lead poisoning. Sometimes, though rarely, it will take place in the nephritic forms. Betterment of vision, however, without improvement of the primary disease is of brief duration. The highest degrees of visual disturbance are seen when the macula or the optic nerve head is markedly implicated or when there is amotio retinæ. Cases of pure albuminuric retinitis seldom produce blindness. If sudden blindness appears, a uræmic amaurosis must be assumed. If a copious hemorrhage takes place in the macular region, where it develops slowly, atrophy of the optic disk generally ensues. If sight grows worse in spells,

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[1] Loco citato.
[2] Ziemssen's Handbuch, 1875
[3] Loco citato.
[4] Loco citato.

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or at first suddenly and then gradually lessens, amotio retinæ must be thought of, especially if vitreous opacities forbid an ophthalmoscopic examination, as is generally the case.

Peripheral vision is usually good. Contraction of the visual field, almost without exception, is seen only with amotio retinæ. I have never seen concentric limitation of the field nor visus interruptus, as Schlesinger has described.[1] Central scotoma is usually found when there are hemorrhages in the macula. Leber has described a paracentral ring-scotoma.[2]

As has been stated by Förster,[3] the light-sense is ordinarily but little altered. The same is the case with the color-sense; yet, according to Galezowski,[4] it may be disturbed where there is a large central scotoma. Schnabel[5] has seen blue-yellow blindness in one instance; Simon[6] has described a case in which there was typical violet blindness, and thinks that this symptom is of no rare occurrence, and that it is characteristic of albuminuric retinitis.

We find the most pronounced retinal changes in the neighborhood of the optic nerve head just as far as the ophthalmoscopically observed peripapillary zone extends. They never reach the ora serrata, and but seldom extend as far forward as the equator. Even macroscopically the swelling and the opaqueness of this zone, as well as the nodulated and wavy condition of the outer surface, can be discerned.

The limitans interna is irregularly thickened, often dim, and sometimes shows tears through which blood from the hemorrhages in the fibre-layer has entered the vitreous. The nerve-fibre layer is thickened and swollen, while its supporting fibres appear hypertrophied and partly degenerated. It is filled with round cells, and, in common with the other layers, is pervaded by a fluid that is rich in fibrin and albumen. Its veins and capillaries are enlarged, and at times new blood-vessels can be seen. In this layer the striped, fusiform, and flame-shaped hemorrhages are situated. The nerve-fibres themselves are hyperplastic and sclerosed. The enlargements are either uniformly cylindrical or fusiform, or appear varicose or club-like. In this latter type they resemble ganglionic cells so closely that by their discoverers, Zenker[7] and Virchow,[8] they were considered to be sclerosed ganglionic cells. Heinrich Müller,[9] however, demonstrated their real character. All these enlargements and the thickened nerve-fibres themselves appear as if filled with a finely granular substance or with granules or minute drops of fat. The enlargements are more frequent in parts, producing localized swellings of the retina. In the ophthalmoscopic picture they appear as shining white plaques or foci of degeneration situated in the peripapillary zone.

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[1] Inaugural Dissertation, Berlin, 1884.
[2] Graefe und Saemisch, Handbuch der gesammten Augenheilkunde.
[3] Zeitschrift für Augenheilkunde, 1872.
[4] Chromatoscopie, Paris, 1867.
[5] Berlin, 1883.
[6] Centralblatt für Augenheilkunde, 1894.
[7] Loco citato.
[8] Loco citato.
[9] Loco citato.

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The ganglionic cell layer is moist and full of round cells. Both granular layers are thickened by hypertrophy, especially the external one, which gives a wavy appearance to the outer retinal surface. These layers often contain a large number of fatty granular cells that are generally round or oval, though occasionally irregular in shape. Their color is slightly yellowish. The aggregation of these cells in certain parts of the granular layers also produces the white or yellowish larger plaques or foci of degeneration which are seen in the peripapillary zone. In rare cases they may be distinguished from the white plaques in the nerve-fibre layer by their yellowish tint. The supporting fibres in the intergranular layer are also hypertrophied, bringing their net-work into better view. In this layer smaller and larger colloid bodies without structure can be seen. I consider these to be due to coagulation of the fibrinous and albuminous fluid which pervades all the layers. In hardened specimens they sometimes exhibit a granular, a fibrillar, or a reticular structure. In these three layers, particularly in the intergranular one, large round, oval, or polymorphous hemorrhages are usually situated.

By the waviness of the outer retinal surface and by the protrusion of the lengthened Müller's supporting fibres the rods and cones are considerably altered. They are pushed from their normal position; in places they are destroyed by pressure and their shape is altered. The radiary fibres (Müller's) appear elongated, thickened, and sclerosed, and in some places they are filled with fat granules or drops. This fatty degeneration of their inner extremities produces the well-known star-figure around the macula seen ophthalmoscopically.

The pigment epithelium also disappears in places from the pressure, or it is dislocated and changed.

The most important changes are those found in the blood-vessels, especially in the arteries. In 1857 Heinrich Müller saw a case of Bright's disease in which the blood-vessels, especially those of the chorioid, were narrowed by proliferation and fatty degeneration of their endothelium. Leber[2] found in albuminuric retinitis that the walls of the arteries were changed into a homogeneous yellow shining tube with narrow lumen; this was the case especially with the small arteries and capillaries, while the larger arteries showed no sclerosis, but merely moderately thickened walls. The work of Duke Karl Theodor is of especial importance concerning this point.[3] The main factor in the pathogenesis of albuminuric retinitis he considers to be an arteritic process of all the blood-vessels of the eye, either an endo- or a meso- or a periarteritis, with narrowing of the lumen, especially of the smaller vessels. This explains the inflammatory and degenerative processes and the hemorrhages in the retina and chorioid. These alterations appear sooner in the chorioid, and are more severe in this situa-

[1] Würzburger Verhandlungen.
[2] Loco citato.
[3] Pathologisches Anstalt der Augenheilkunde bei Nierenleiden, 1887.

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tion than in the retina. He considers that the chorioid possesses a separate system of capillary blood-vessels, and that the retinal arteries are terminal ones, which are especially favorable to the development of the affection. These circulatory conditions in the retina and chorioid, which somewhat resemble those found in the kidneys, bring about a condition, especially after the area of the renal blood-vessels has been obliterated and the blood-pressure is accordingly increased, in which the blood-stream is retarded in these two membranes and the toxic substance in the blood remains longer in contact with the blood-vessel walls and hence causes their diseased condition. When the lamina are considerably reduced or obliterated, a dropsical necrosis of the nerve-fibres results, as well as an extravasation of the constituents of the blood and hemorrhages, occurring the more readily since the same changes have developed earlier and to a higher degree in the chorioid. In consequence, the arterial circle of Zinn can no longer act as a safety-valve against the increased pressure in the retinal arteries. Another point which he considers to be of great importance, especially in the production of retinal changes in the peripapillary zone, is that in the physiological excavation the retinal blood-vessels bend abruptly (almost at right angles), thus breaking the blood-stream and giving rise to regressive metamorphoses in the blood-vessel walls themselves. Michel[1] has found hyaline degeneration of the walls of the arteries in the retina, chorioid, and kidneys, and considers these changes to be all due to a common cause.

Concerning the optic nerve head, I know of no other description than that of Leber,[2]—namely, swelling dependent upon the albuminous and fibrinous fluid, which is found also in the retina, accompanied by round-cell infiltration, hypertrophy of the interstitial connective tissue, gray degeneration, and the appearance of numerous corpora amylacea.

According to Poncet,[3] hemorrhages and plaques of fatty degeneration, which are but rarely seen with the ophthalmoscope, are found in the papilla. The chorioid usually participates in the disease, and the same blood-vessel alterations as are seen in the retina—namely, sclerosis and fatty degeneration of the epithelium—are found. Sometimes chorioiditis and colloid excrescences of the lamina vitrea can be seen. (Müller.) In the vitreous humor diffuse or circumscribed opacities can sometimes be determined ophthalmoscopically. At times there is an increased number of cellular elements, and, finally, hemorrhages are not infrequently found.

Denissenko has determined the presence of inflammatory changes in the cornea.

Albuminuric retinitis never appears before the kidney-disease, but is always found in the later stages of the disease, and sometimes in the latest. It almost always attacks both eyes, yet not at the same time nor with equal intensity. Monocular affections have been seen by Cheatham, Bull, and Ivert.[4] Very often it is the first symptom of the serious disease of which

[1] Loco citato.
[2] Loco citato.
[3] Congrès d'Ophtalmologie, 1880.
[4] Loco citato.

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the patient becomes aware. The ophthalmoscopic appearance is such as to lead to a proper diagnosis by the physician.

The course of the disease is eminently chronic, and in typical cases three stages can be generally distinguished. Vision is extremely variable. If the improvement does not depend on a betterment in the primary disease, it does not last.

Uræmic attacks, often combined with momentary or lasting uræmic amaurosis, may happen. The termination in a cure with a restitution of vision is possible, and all the pathological alterations in the fundus may partially or entirely disappear. Such favorable cases are often observed in those forms of albuminuric retinitis that are based on an acute nephritis, as in pregnancy, childbirth, the acute exanthemata, poisons, etc. Cases dependent on chronic nephritis or shrinking kidney are cured probably in extremely rare instances only. Adamük[1] has seen such a case of cure. A partial but lasting cure has been seen by Steffan[2] and others. In one case Galezowski has seen an albuminuric retinitis terminate in hemorrhagic glaucoma.

Albuminuric retinitis may occur in all forms of kidney-disease which produce albuminuria. It is most frequent in shrinking kidney or granular kidney or in primary atrophy of the kidney. It is rarely found in chronic nephritis or in cases with the so-called large white kidney; more rarely still is it seen in acute nephritis as observed during pregnancy, in childbirth, after the acute exanthemata, especially scarlet fever; and less frequently in variola and measles, erysipelas, carbuncle, typhus, diphtheria, intermittent fever, or intoxication with lead or cantharides. Chronic nephritis is usually thought to be due to repeated colds or drenchings,[3] to chronic suppurations, as in caries and necrosis, to different so-called dyscrasiæ, as tuberculosis, syphilis, and scrofula, and to the abuse of alcohol and mercury.

According to Beckmann, Traube, Alexander,[4] and others, an amyloid degeneration of the kidneys in syphilis, tuberculosis, or suppuration of bones may be followed in its later stages by albuminuric retinitis and diabetes. Traube[5] considers hypertrophy of the heart as the pathogenetic factor in the production of albuminuric retinitis. Leber[6] has shown that this is improbable. Von Graefe and Leber were the first who stated that the uræmic condition of the blood is the cause of this retinitis, and this opinion is probably shared by most ophthalmologists. Landouzy's opinion that albuminuric retinitis is observed in most cases of kidney-disease has long since been refuted. Galezowski says it occurs in thirty-three per cent.; Lécorché, twenty-one per cent.; Lebert, twenty per cent.; Frerichs and Miles, thirteen per cent.; Bright and Barlow, ten per cent.; Wegner, nine per cent.; and Schweigger, from six to seven per cent. I think we shall not be very far astray in accepting from nine to ten per cent. as the average.

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[1] Centralblatt für Augenheilkunde, 1888.
[2] Jahresbericht, 1873.
[3] Niemeyer, Lehrbuch.
[4] Loco citato.
[5] Loco citato.
[6] Loco citato.

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The diagnosis of albuminuric retinitis, as a rule, is not difficult, since there is probably no other form of retinitis that presents so characteristic a picture. The less the typical symptoms are developed the more difficult becomes the diagnosis. Moreover, if the albuminuric retinitis appears with the ophthalmoscopic picture of a hemorrhagic one, or if it manifests itself as hemorrhages into the retina, or a papillo-retinitis, or a typical papillitis without any affection of the retina, the diagnosis by means of the ophthalmoscope alone is rendered impossible. Since, however, we know that most of these signs in the fundus may be wanting and others (papillitis) may be added, it is imperative for the conscientious physician to examine the urine for albumen in every case of retinitis, neuro-retinitis, or papillitis.

In making a differential diagnosis concerning the character of the several white plaques in the fundus, it should be remembered that small shining white or pure white ones covering some retinal blood-vessels may safely be looked upon as degenerated nerve-fibres, while larger areas of a dull-white or yellowish color over which some blood-vessels are passing must be considered as foci of fatty degeneration that are located in the granular layers.

The prognosis of albuminuric retinitis is always very serious. Aside from the cases that are due to an acute nephritis, as in pregnancy, the acute exanthemata, etc., in which a cure is often observed, it will be found that in almost all instances death must be expected in a few months or years. In consequence, therefore, the appearance of the retinal affection in renal disease is of the gravest prognostic importance. Moreover, since it is known that blindness almost never follows albuminuric retinitis, while an early death nearly always takes place, it must be agreed that the retinal disease becomes of graver prognosis in regard to life than it does in reference to vision.

Concerning the duration of life after the albuminuric retinitis has been found, Baroness Possauer[1] has collected the cases out of sixty-seven thousand cases from the Zurich clinic and the private practice of Professor Haab. The following data are taken from her work. Men of the poorer classes coming to the clinic die within two years,—that is, their percentage of death is one hundred; while among women of the same class the percentage is only sixty-eight. Of the private patients possessed of means, the death-rate is fifty-nine per cent. for men and fifty-three per cent. for women. The longest period of life after the retinitis had been diagnosed was six years for the clinical cases and eleven years for the private ones. This great difference in the prognosis between the poor and the rich is, of course, due to the fact that the first live under much more unfavorable conditions, cannot follow the advice of their physicians as regards diet, and seldom relinquish the use of bad alcoholic liquors, which are worse than larger quantities of good quality. It is evident that men suffer more under all these noxious influences than women, because the former must provide for the

[1] Inaugural Dissertation, Zurich, 1894.

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maintenance of the family. The figures given here show, however, that, in spite of the gravity of this disease, not only can the physician ease the sufferings of these patients, but he can also prolong their lives, if they follow directions.

In the way of treatment of the eye-affection, local hygiene and protection by means of shading glasses are all that can be suggested. Arlt's ointment, iodide of potassium ointment, or tincture of iodine applied to the temples can probably do no harm, though they can be used only as a placebo. Methodical treatment with eserine as recommended by Knies[1] I have never tried.

XXX. STRIATED RETINITIS, PUNCTATE RETINITIS, CIRCINATE RETINITIS (RETINITIS STRIATA, RETINITIS PUNCTATA ALBESCENS, RETINITIS CIRCINATA).

Striated Retinitis.—Under the name of striated retinitis, Nagel, of Tübingen, has for many years described certain forms of retinal changes which consist of white, arborescent, retrovascular stripes that appear in a greater part of the fundus.

As I have no knowledge of a communication by Nagel on this subject, and as I have only very recently and very cursorily had occasion to observe such a case, I shall make use of the description by Kumadjin Iosiaki Onisi,[2] which was authorized by Nagel. According to this author, the first two cases were seen and pictured by von Jaeger.[3] Both of these appeared in young men and were monolateral in type. No etiological factors could be found. Hirschberg[4] has described a binocular case which was associated with retinal detachment. Onisi[5] describes five instances seen in Nagel's clinic. The first was in a student; the second in an individual fifteen years of age, in whom the affection had been preceded by a slight injury; the third in a thirty-five-year-old woman who had taken cold; the fourth in a twenty-one-year-old woman who had a cysticercus in her eye; and the fifth in a twenty-nine-year-old woman. Kaspar and Krüger[6] have related a case found in Saemisch's clinic which occurred in a forty-one-year-old microcephalic man.

The characteristic changes are light stripes or bands, that always lie behind the retinal vessels and in front of the pigment epithelium. Sometimes they are very fine, while again they may be three or four times broader than the retinal veins. Their course is straight, arched, or wave-like. Their color is shining white, yellowish, bluish, or greenish white, and some of them are bordered with pigment. Their edges are usually sharp and distinct. The long stripes generally run like radii from the optic nerve

[1] Loco citato.
[2] Inaugural Dissertation, Tübingen, 1890.
[3] Beiträge zur Pathologie des Auges, 1870, Atlas, Tafel XXXIII.
[4] Berliner klinische Wochenschrift, 1870.
[5] Loco citato, 1890.
[6] Festschrift für Helmholtz, 1891.

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head to the periphery of the retina, yet there are cases in which they traverse the fundus in all directions or extend from large circles situated around the optic nerve head. Near the optic disk their distinctness and sharpness of outline decrease, and they may terminate in that situation as a sharp point or a hook, or there may be a fork-like division or a loop. Towards the periphery most of the stripes become split and generally pass over into whitish-gray, sometimes pigmented, foci or localized retinal detachments. When they do not end in these patches, they recurrently branch dichotomously. Sometimes a stripe may be divided into two diverging branches situated close to the optic disk. The stripes may have localized enlargements. They may branch off once or several times. Star-shaped figures may result from their divisions and anastomoses. Sometimes they are combined with white points and dots.

The optic nerve head is normal or pale, and its contours are sometimes indistinct. At times the retinal vessels are tortuous, and the retina may become diffusely opaque around the optic disk. There may be white or yellow dots and points or stripes and pigment accumulations in the macular region. The vitreous humor may be dim.

Visual acuity is considerably reduced, though light- and color-sense suffer rarely. The visual field sometimes shows defects, but blindness does not generally result.

The course of the affection is chronic. Ordinarily it has existed for years before being observed, and some cases have been under observation for years without showing any change.

The etiology and pathogenesis are unknown.

As a rule, the disease attacks young people, all the cases observed having appeared between the fifteenth and forty-first years of life. Sometimes an injury to the eye has occurred. In one case there was a cyst, perhaps a cysticercus, but it is not known whether these were among the causes of the condition.

In the differential diagnosis one might think mostly of proliferating retinitis (Manz), yet in this affection the white connective-tissue bands lie in front of the vessels. It cannot be confounded with the striped and band-shaped areas that are frequently seen after the readaptation of detached retinas.

Therapeutic measures are useless.

Punctate Retinitis.—Mooren gave this name to a form of retinitis which he saw in a thirty-year-old man. He found several hundreds of white spots equally distributed over the whole eye-ground. Unfortunately, his description of this case was so brief and defective that very different forms of affections were described under this name, until Fuchs[1] studied this form of retinitis somewhat more accurately.

In this form of retinal inflammation the spots are small, white or yel-

[1] Archiv für Augenheilkunde, Bd. xxxix.

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lowish white in color, without pigment border, and are equally distributed over the fundus. Generally they are several hundred in number. They never coalesce. The fovea is ordinarily free from the spots.

The affection is found in young individuals, and is often present in members of one family. Consanguinity in the parents can frequently be determined.

The patients usually suffer from day-blindness. Their central vision is reduced, and there is marked concentric limitation of the visual fields. The day-blindness has generally existed since early childhood. From this it may be concluded either that the disease is congenital in type or that it develops in earliest infancy.

The external appearance of the eyes is normal and the media are clear. Small, dull-white points, which are more numerous around the optic nerve head and the macula, can be found all over the fundus. Their relation to the blood-vessels is not always the same. There are no other pathological fundus-changes. Central vision is reduced, while peripheral vision is normal.

There is no night-blindness. The affection begins between the fifteenth and twentieth years of life. The number of reported cases is as yet too small to allow an accurate picture of the affection to be formulated.

Circinate Retinitis.—Under this name Fuchs[1] has described as a separate form of disease a rare variety of retinitis, the chief characteristic of which consists in an alteration in the macula, that at a certain distance is surrounded by a zone of white spots. Among seventy thousand patients he observed this affection in twelve cases, and but eight of these were typical. Ten women and two men were affected, the youngest of whom was thirty-eight and the oldest seventy-five years of age. The average age was sixty years. The affection was binocular in five cases and monocular in seven. De Wecker[2] has seen the same affection only fifteen times among one hundred and fifty thousand patients. He thinks that the condition described by Fuchs is not a special disease, but must be considered as a fatty degeneration following hemorrhagic retinitis. De Wecker and Masselon have always found this affection associated with hemorrhages in the retina, and are convinced that the hemorrhages have preceded the appearance of the white spots. In some cases they saw the foci of degeneration appear in the same spot where there had been a previous hemorrhage. In old cases they often noticed small hemorrhages situated near the white spots. These facts have convinced de Wecker that he had to deal with a condition of fatty degeneration. He believes that at first the vessels surrounding the macula are diseased. In the course of years this ring gradually increases towards the large temporal branches, and at times extends even farther. He has seen a larger number of atypical cases, and infrequently

[1] Archiv für Ophthalmologie, Bd. xxxix., 1893.
[2] Annales d'Oculistique, Bd. 1894.

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has found the spots forming a straight line (once even on the nasal side of the nerve head) instead of a ring. In all his cases the spots were bordered by pigment. He determined that the affection always started from the macula and increased centrifugally. The macula was altered. In new cases he determined the presence of punctiform hemorrhages in this region, the larger ones being seen only in the older cases. He cites the case of a sixty-year-old diabetic subject who had circinate retinitis typically developed in one eye and hemorrhagic retinitis in the other as proof of the connection between the two affections. He never found the spots prominent, nor did he ever see them disappear, as Fuchs has stated has been his experience. De Wecker gives extended descriptions of several cases, especially of one which he observed during a period of twenty years, and which had also been seen by Rothmund, Becker, Donders, Bowman, and Knapp. Holmes Spicer[1] has seen a man, seventy-three years old, with small hemorrhages in the right eye and a typical picture of circinate retinitis in the left.

According to Fuchs, circinate retinitis is characterized ophthalmoscopically by a gray or grayish-yellow opacity situated in the macula and its neighborhood, which at a certain distance is surrounded by a zone that is composed of smaller and larger white spots. The macular opacity is seen in recent cases only as a small, well-defined spot, which is always larger than the optic disk. This dimness lies behind the vessels. In severe cases this portion of the retina may be swollen and there may be small hemorrhages in the macular region, and, very rarely, pigment-spots can be recognized in old cases. The ring of spots is separated from the macula by a zone of tissue that is but slightly altered. In some types the ring may include the optic nerve head. It is seldom closed, being usually open on the nasal and temporal sides. In recent cases it is but partially developed. The ring of spots does not form a circle, but assumes the shape of a horizontal ellipse. Its diameter is two or three times that of the optic disk. The large temporal vessels generally pass by at its periphery without touching it, and only the macular vessels cross it. In some cases, however, the temporal vessels may come together with its upper and its lower edge, and then some spots can be seen to lie along these vessels and to produce arborescent figures. The ring consists chiefly of milk-white spots of a dull color, well defined, and without pigment borders. Sometimes they coalesce, forming lobulated figures. These again may be united to larger white spots, resulting in the formation of a large continuous white area, the lobulated margin alone showing its origin. Near such large areas there are some small white spots. In rare instances these large lobulated areas appear from the beginning.

The retinal vessels pass over these spots, although the latter may protrude from the retinal surface. A zone of normal red fundus remains between the macula and the ring of spots, yet even when this portion is

[1] Ophthalmic Review, 1894.

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carefully examined it will be usually found to be slightly opaque. The optic disk may be normal or somewhat hyperæmic and indistinct in outline. In recent cases the blood-vessels are generally normal. Rarely they are somewhat broader and tortuous. In some instances hemorrhages, which are ordinarily minute and punctiform, are found. The remainder of the fundus is either normal or shows changes, such as, for instance, colloid excrescences of the chorioid. In recent cases the vitreous humor is transparent.

According to Fuchs, the visual disturbance develops gradually, the patients being generally unable to state when any deterioration of vision began. It consists in a diminution of central vision with a limitation of the visual field. Visual acuity is greatly and permanently impaired, being usually reduced to the ability to see to count fingers at from one to three metres' distance. A central scotoma of a diameter of from ten to twenty degrees always has been present in the cases reported. In recent cases the peripheral field is normal; in old ones it is diminished. The light-sense seems unaltered. Metamorphopsia, night-blindness, and day-blindness are never seen. Photopsiæ have been complained of but once.

The white spots cannot be considered as evidences of sclerosis of the nerve-fibres, because the vessels would then appear partly covered. Accumulations of round cells are not probable, since there are no marked inflammatory symptoms. Fatty degeneration as the result of hemorrhages, according to de Wecker and Masselon, might, in accordance with Fuchs, develop in spots in the retina without preceding hemorrhages, and it cannot be excluded as a cause of the white spots. Fibrinous exudation into the retinal tissue and coagulation with the formation of lump-like masses may explain these spots, as Fuchs has assumed. The regular circummacular localization of the zone of spots shows their causal connection with the blood-vessels situated around the macula.

The course of the affection, as observed by Fuchs in one case which lasted seven years, and by de Wecker in one that existed for twenty years, is chronic. The ophthalmoscopic appearance often remains unchanged for years. According to de Wecker, new hemorrhages and fresh white spots may be formed. Their disappearance is stated by Fuchs to be observed best in the ring of white spots; this, however, is denied by de Wecker. These areas crumble to pieces and form smaller ones. Their outlines become indistinct, gradually their white color is lost, and some may even exhibit crystals of cholesterin.

Fuchs has seen a case in which the macular spot as well as the zone of white spots disappeared in four years. In other instances considerable pigment degeneration has been observed. In yet other cases the exudation does not disappear, and gradually the retina becomes enormously thickened. The macular spot and the zone of white spots are then changed into dirty yellow-white, protruding, connective-tissue-like masses. Hemorrhages, which are generally absent or very small in recent cases, appear more frequently and are larger in the older types. They sometimes show the well-known

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whitish discoloration. Later dust-like or larger vitreous opacities may appear. Fuchs has twice seen retinal detachment and once a broad posterior synechia. He has not found any definite particulars regarding the etiology of the condition, although he has looked particularly in every case for evidences of syphilis, and has examined the urine. The physical examination, never omitted, revealed once a moderate degree of arterial sclerosis, and twice the same condition combined with hypertrophy of the left ventricle. Rigid blood-vessels were almost always present, the patients being nearly all over sixty years old.

An important point is that the affection preferably attacks women, especially those who are advanced in life, making it seem as if senile processes are responsible for the condition. In making the differential diagnosis diabetic retinitis might be thought of. According to Fuchs, however, diabetes never produces the formation of a perfect ring of spots, and in his cases diabetes was absent. In addition, de Wecker has described a case of circinate retinitis found in a diabetic. The same holds good for albuminuric retinitis. Senile changes in the macula, as described by Waren Tay, Hutchinson, Hirschberg, and others, can hardly be confounded with circinate retinitis. In these the spots are never pure white in color, but are yellow in tint, being indistinctly outlined and often bordered with pigment. They never form larger white areas by confluence, and are most numerous at the macula itself, which region is always free in circinate retinitis. The affection is always binocular, and vision is but little reduced.

Retinitis punctata albescens is found in younger individuals, and is accompanied by concentric limitation of the visual field with day-blindness. The picture is never that of typical circinate retinitis. Prognosis is bad, and therapeutic measures are useless. Total blindness seems, however, never to result from circinate retinitis.

RETINAL DETACHMENT (AMOTIO RETINÆ).

This condition was well known in preophthalmoscopic times, and in the last century anatomical examinations of such eyes were made, as, for instance, in 1722 by de Saint-Yves.[1] In 1853 Coccius[2] gave the first ophthalmoscopic description, followed in the same year by von Trigs.[3] In 1854 von Graefe[4] assumed a hemorrhagic cause, basing his conclusion upon the sudden appearance of the condition.

The manner in which retinal detachment takes place is to this day not fully explained for all cases. There exist several opposing theories, each based on more or less valuable proof. The first theory is that of secretion or exudation, conceived by Arlt, although Wardrop, in preophthalmoscopic times, had tried to offer a similar explanation. Later Schmidt-Rimpler,

[1] Traité des Maladies des Yeux.
[2] Augenspiegel.
[3] Nederlandische Lancet.
[4] Archiv für Augenheilkunde, Bd. i.

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Boucheron, and others have adopted this theory. Here it is assumed that the subretinal fluid comes from the chorioid, and that this secretion presses the retina against the vitreous humor, thus bringing about its gradual absorption. Later, aside from the subretinal chorioidal exudation, a process of shrinking in the vitreous humor, due to a contemporaneous cyclitic process with a plastic, fibrinous, and shrinking exudation into the anterior parts of the humor, has been assumed.

This exudation or secretion theory may explain slowly developing cases of retinal detachment, and is, even to-day, probably correct for some types that are combined with retinitis, especially the albuminuric forms, glioma, sarcoma, and subretinal cysticercus. It does not explain, however, the sudden so-called idiopathic retinal detachments, since it is inconceivable that so large an exudation could be thus formed in a few moments; and even if this were possible, and this exudation could overcome the normal pressure of the vitreous humor and press the retina inward, the intra-ocular tension must needs be enormously increased, and that is just the condition that is hardly ever found in retinal detachment. On the contrary, the rule is that intra-ocular tension is diminished.

The second theory is that of extravasation or hemorrhages. This was first enunciated and then abandoned as a general explanation by von Graefe, and was partly supported by Horner. On account of the suddenness of the appearance of the retinal detachment in some cases, the former thought the only explanation to be that of a sudden hemorrhage which took place from the chorioid. However, in some cases the same reasons hold good against this as against the sudden exudation theory, particularly the absence of hypertonus. It is, however, correct in some types, especially after injuries with loss of vitreous humor, after operations, in some forms of retinitis, generally the hemorrhagic types, and in hemorrhagic glaucoma.

The third theory is known as the mechanical or distention theory. This was also originated by von Graefe, and was formulated to explain retinal detachment in highly myopic eyes, in which it is so frequently observed. Von Graefe, and later Donders, explained this variety of detachment by the elongation of the antero-posterior axis of the eyeball, assisted by the hyperæmia which accompanies posterior sclerectasia. This theory, too, was found inapplicable to all cases, it being difficult to understand why in these instances the retina should be less elastic than the other membranes, and, if this were so, why it should not rupture, instead of becoming detached. The discoveries of Iwanoff were of the greatest importance for this and the following theories. He[1] found in highly myopic eyes at the posterior pole a detachment of the vitreous humor, and his explanation of this fact was that this humor could not increase in volume as rapidly as the posterior part of the sclerotic was distended. In consequence, serum would have to fill this gap. At the edge of the detachment he determined that the

[1] Loco citato.

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vitreous humor was attached to the retina. Iwanoff thinks that the exudation behind the vitreous humor finally lacks room and must either filter into the humor itself or pass backward through the retina and thus lift this membrane from the chorioid. This explanation, however, is incorrect and against all physical laws, since the fluid between the vitreous humor and the retina must exert an equal pressure in all directions, and therefore can press the retina more firmly against the chorioid, but can never detach it from this membrane. On this discovery of Iwanoff, de Wecker[1] based his modification of the distention theory by the assumption of a rupture of the retina. He sometimes found in highly myopic eyes spontaneous ruptures in the retina, which he explained by assuming that they originated from the pressure of the preretinal fluid while the eyeball became stretched, stating that this fluid would pass behind the retina through the rupture, thus causing the retina to become detached.

Before leaving the distention theory we must remark that both this and the secretion or exudation theory have still their adherents among prominent ophthalmologists, and certainly justly so for some forms of retinal detachment. Thus, Schweigger[2] states that detachment of the retina is not always due to one and the same cause, but may develop in different ways. The most frequent form, that in myopic eyes, he explains by an amount of stretching of the retina equal to that of the sclerotic and the chorioid, and by the consequent rupturing of this delicate tissue upon the slightest provocation. If, as is the rule in high-grade myopia, the vitreous humor is liquefied at the same time, the vitreous fluid oozes through the rupture and is accumulated between the retina and the chorioid, and the detachment results. Schweigger has observed sometimes that two or three days elapse after the rupture has taken place before the patient notices any diminution of vision. He has also, exceptionally, seen rupture and detachment of the retina in eyes which were not myopic. A second group of detachments Schweigger calls the exudative ones. To this type belong the detachments seen in albuminuric retinitis and those which are occasionally observed during affections of the orbit. Schweigger also thinks that retinal detachment may be caused by inflammatory exudations from the chorioid without visible symptoms of inflammation. Schnabel considers that idiopathic detachments are due to a secretory neurosis.

In opposition to the secretion, exudation, extravasation, and distention theories, Leber and his pupil Nordenson have brought forward the theory of retraction or shrinkage, which has been confirmed by many other observers and is at present almost universally accepted. In reality, however, H. Müller originated the retraction theory, though he was greatly biassed by the secretion theory. He first announced it as his opinion that detachment of the retina could result from shrinkage of the vitreous humor, and

[1] Loco citato.

[2] Bericht des zwanzigsten Versammlung der ophthalmologische Gesellschaft, Heidelberg.

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he based this opinion on the examination of three eyeballs that were enucleated by von Graefe for irido-chorioiditis. Stellwag von Carion[1] was, however, the first to call attention to the fibrillar condition of the vitreous humor in such cases and to point out its importance in retinal detachment. Moreover, he showed how untenable were the previous theories in explanation of most cases. To Leber and Nordenson we owe much for having given the retraction theory a firm foundation by means of numerous ophthalmoscopic observations, anatomical examinations, and experiments. Leber considers his opinion as proved,—namely, that retinal detachment, with the exception of a few rare forms, is always produced, not by a primary chorioidal exudation, but by traction of the shrinking vitreous humor. From his and Nordenson's investigations there remains no doubt that retinal detachments which can be diagnosticated with the ophthalmoscope originate in this manner. These investigations make plain the shrinking of the vitreous humor as well as the strange spontaneous ruptures of the retina, and explain why it is impossible to see this process by ophthalmoscopic examination alone.

Without losing its transparency, the vitreous humor gradually assumes a fibrillary structure during its condensation and is contracted to a smaller, steadily diminishing volume, the space thus resulting being filled by a serous fluid. The anterior part of the retina which adheres to the condensed vitreous humor is now in a condition to be pulled inward. This condensation of the vitreous humor, to which, in some cases, processes of proliferation may be added, is due to a chronic inflammation of the chorioid. The traction produced by the shrinking vitreous humor may lead to a tearing of the retina. The serous fluid between the vitreous humor and the retina enters through this tear into the subretinal space, the more rapidly the larger the tear. In this manner a detachment may develop suddenly and without any increase of intra-ocular pressure, since the transudation in the vitreous space simply changes the locality of the membrane. This is certainly the explanation of sudden detachments with unchanged tension.

When the detachment develops slowly, a primary rupture of the retina is not necessary, as the retina may be slowly drawn away from the chorioid while at the same time fluid is collected behind it.

In opposition to the retraction theory of Leber-Nordenson, Raehlmann has advanced what is known as the diffusion theory.[2] He does not agree that the anatomical data of Leber-Nordenson are convincing proofs, since the examination of the eyes studied was mostly made a long time after the detachment had taken place, it therefore being impossible to decide whether the changes in the vitreous humor preceded the detachment or were developed later. He further calls attention to the fact that high-grade, even scar-like, shrinking of the vitreous humor has been often observed without reti-

[1] Loco citato.

[2] Archiv für Augenheilkunde, Bd. xxvii., 1893.

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nal detachment, and that, on the other hand, retinal detachment frequently develops without any preceding shrinkage of the vitreous humor, and even, on the contrary, when synchysis of the vitreous humor has been observed. He has repeated Leber's methods of production of artificial detachment in animals, and denies that they constitute a proof of the retraction theory. He explains them by his diffusion theory, since the processes are not only of a chemical but also especially of a physical type. Even the retinal rupture, so necessary for the Leber-Nordenson theory, he does not accept as argument, since Leber and Nordenson themselves have acknowledged that there are a great many cases seen without it. On the other hand, detachment does not necessarily occur even with the highest degrees of myopia. Furthermore, he does not believe that the shrinking of the vitreous humor alone can explain the retinal tear, while he thinks that his diffusion theory does it without any difficulty.

It is well known that the process of diffusion presumes differences in tension in two fluids, one of which is in front of and the other behind an animal membrane. These conditions exist when the retina is detached. Behind the retina there is a relatively albuminous fluid, while in front of it in the vitreous chamber there is a fluid that contains but little albumen, the diffusion taking place the more rapidly the greater the difference in density of the two fluids. As a rule, the pressure is probably much higher in the subretinal space than it is in the vitreous chamber, since the watery fluid is transuded much more quickly and more plentifully into the subretinal space than the albuminous subretinal fluid is into the vitreous space. In consequence, the eyeball will usually be found to be soft, and there will be a relatively deep anterior chamber, while the retina is more or less tense. When during this process of tension the force of the subretinal fluid becomes too strong for the power of resistance of the retina, the latter is torn.

Raehlmann contends against the retraction theory that by it a reapplication to the chorioid cannot be explained, whether this results from spontaneous cure, as is so often seen, or from therapeutic measures; while according to the diffusion theory this method of reposition is easily understood by assuming that after the equilibrium between the two fluids and normal nutrition has been re-established the subretinal fluid is absorbed. For this and the other reasons it is clear that Raehlmann's diffusion theory, like that of Leber and Nordenson, assumes as the last cause of retinal detachment an inflammation of the chorioid, with the difference that the chorioidal exudation is collected under the retina, instead of entering the vitreous chamber.

Retinal detachment is materially aided by a system of fissures in the retina which are produced by the process of diffusion. This system of fissures in the retina when it becomes detached, as observed and pictured by Raehlmann, is said to be similar to the findings of Iwanoff[1] and Dennis-

[1] Archiv für Ophthalmologie, Bd. xv.

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senko.[1] It is also found in the eyes of animals in which the vitreous humor has been artificially detached by the injection of salt solution. It seems, therefore, that this system of fissures plays an important part in the process of diffusion and in the pathogenesis of detachment of the retina. Rupture of the retina, of which Raehlmann could not always convince himself, and which is the condition necessary for the Leber-Nordenson theory, is for him as irrelevant as the shrinkage of the vitreous.

When Raehlmann's statements, which we have here reported extensively on account of the importance of this question, are carefully considered, it must be confessed that the diffusion theory is a very attractive one and explains much better many more points than do the other theories. Its weak point, however, is the difficulty in explaining the sudden appearance of detachment. We can hardly understand how the process of diffusion could take place so rapidly that a large detachment could be formed in a few minutes, or even instantly, a condition that can be easily explained by the retraction theory.

If, without having studied this affection of the retina more particularly, I may be allowed, from having more or less carefully observed several hundreds of clinical cases of detachment, to pass judgment in this matter, I will state it as my opinion that for the vast majority of ophthalmoscopically demonstrable retinal detachments the retraction and diffusion theories alone can be considered, while the secretion, exudation, extravasation, and distention theories can be applied only to exceptional cases.

Some further theories concerning detachment we shall merely mention. The theory of Rava[2] assumes a circulatory disturbance in the chorioid, due to passive hyperæmia from atrophy of the walls of the blood-vessels. Here an affection of the vitreous humor is unnecessary for the development of detachment, since this, as well as mechanical pressure from the orbit or stasis in the ophthalmic veins, may follow. In highly myopic eyes, too, this author sees the retinal detachment produced by congestion in the chorioid, and not by liquefaction of the vitreous humor. Unterharnscheidt[3] thinks that detachment in myopic eyes is produced by a sudden relaxation of the ciliary muscle, with consequent decrease of the vitreous tension. This, he says, cannot be equalized by an equivalent contraction of the sclerotic, thus allowing the production of the retinal detachment. Kuhnt considers detachment in some cases as the result of cystic formation dependent upon senility.

Even externally, certain symptoms of retinal detachment can be usually found. There is especially a reduction of intra-ocular tension. It is rarely normal, and extremely seldom is it increased. The anterior chamber is mostly deep. Sometimes it is very deep, and in such cases iridodonesis may be observed. The pupil is wide and the iris is sluggish. Later discolora-

[1] Archiv für Augenheilkunde, Bd. xix.
[2] Annali di Ottalmologia, ix.
[3] Berliner klinische Wochenschrift, 1880.

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tion of the iris, loss of iris-reaction, a wide pupil, and some posterior synechiæ may be added. Finally posterior polar cataract may ensue.

Sometimes in high-grade detachments of the retina a gray reflex can be seen with the naked eye when the light is favorably situated. In very large ones, and especially when there is pus collected behind the retina, the symptoms of the so-called amaurotic cat's eye may be produced. When the pupil is wide and the light is intense it may be possible to observe even the retinal blood-vessels with the naked eye.

Ophthalmoscopically three groups of the condition can be differentiated: 1, very flat ones, of varying extension; 2, partial ones, springing inwardly into the vitreous space; and, 3, total, funnel- or convolvulus-flower-shaped ones.

Those of the first group are often difficult to diagnose ophthalmoscopically unless some fold-like elevations which direct the observer's attention to the interrupted course of the blood-vessels can be recognized. Such folds usually run in an equatorial direction, there being mostly several lying side by side. They appear as white, arch-like stripes, and at their edges the blood-vessels seem bent or cut off. The parallactic movements of the retinal and chorioidal vessels, and the darker color of the blood-vessels, which in larger detachments may become even black, make the detachment plain.

In the second group, instead of the normal red reflex, a shining white, yellowish, bluish, or greenish white portion, which during the movements of the eyes may recurrently appear in the field of view, can be generally seen by mere illumination of the media with the ophthalmoscope. This will be found in a more extended locality. On examining more accurately, this whitish portion will be found to be on a different level from the remainder of the fundus, and it will be recognized by means of the blood-vessels that a detachment of the retina, which shows upon the slightest movement of the eye a wave-like, tremulous motion, is being dealt with. The degree of the detachment may be determined from the difference in focus for the detached and the normal portions of the retina. The detached part may appear flat, hill-like, or abrupt and steep. Large portions, as a rule, will be found detached. Small localized and well-defined detachments are rare. The large majority lie downward; yet it must not be forgotten that those which begin in an upward position easily sink downward, while the retina upward readapts itself to the chorioid, so that if a downward detachment has not been seen in its beginning it becomes impossible to determine whether it originated in an upward position and gradually sank downward or not. Detachments are rare at the macula. The rarest ones are on the medial side of the retina.

Usually one eye only is attacked. When both are affected, which is seldom, but one is involved at a time. The detached retina appears as a trembling, floating membrane which is rarely tense. Sometimes it forms a sac with elevations that overhang the normal retina. In some cases it remains more or less transparent, though in most instances it is dim or

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opaque. Its color depends on its degree of transparency or the color of the subretinal fluid.

When the retina is transparent and the subretinal fluid is clear, the detached portion differs but little from the remainder of the fundus, and can, as a rule, be recognized only by a trembling motion. In other cases the detached part appears gray-white, whitish blue, or green. It may also look like blood. When the detachment lies in the macular region, the macula will be seen as a blood-red spot in a white field similar to that found in embolism of the central retinal artery.

When the retina is opaque, the color of the subretinal fluid cannot be determined. The retina then appears gray-white, and is sometimes strewn with separate nebulous white spots. Hemorrhages and pigment-areas are but rarely found on the detached portions. More frequent are spontaneous ruptures, which generally form arch-like tears with inverted edges. When the tears gape, the chorioid may sometimes be plainly seen through them.

In larger detachments the blood-vessels are of a dark or even black color. They sometimes appear as black dendritic figures, which to the inexperienced may seem like thrombosed vessels. According to Liebreich, this dark color does not depend on a change in the color of the blood, but is an optical phenomenon due to the fact that the vessels absorb more light in transillumination. In the erect image the vessels look thin, because they are seen under a smaller magnifying power. Later disturbances of circulation result, the blood-column may appear broken, and finally thrombosis and shrinkage may occur.

In the third group the fundus loses its normal color and appears in the different shades of a detached retina, as detailed. When the media are clear, a strong convex lens about —25 D. may be used to look into the detached retina as into the funnel of a convolvulus flower, and some parts may be seen protruding, while deep indentations and small recesses may be found between them. In old cases hemorrhages and their derivatives, pigment accumulations, lime deposits, and cholesterin crystals may all be found.

The most common complications are synchysis and opacities of the vitreous humor, these being especially seen in myopic eyes. Alterations in the chorioid and pigment epithelium, which are rarer, usually exist in the neighborhood of the detached part. Partial cataract in the posterior cortex of the lens, or consecutive total cataract, which generally starts from the posterior pole of the lens, can be seen. It is necessary, however, to distinguish between consecutive cataract dependent on the detachment and simple senile cataract or phacoscleroma that has developed in an eye with a previous detachment. This is also true of cases in which a detachment occurs in an eye with senile cataract.

Not infrequently chronic iritis with synechiæ may be found as a further complication. More rarely albuminuric, hemorrhagic, or purulent retinitis may be present.

The amount of visual disturbance is in proportion to the extent and the

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locality of the detachment. The retina corresponding with this defect is often absolutely void of light-sensation. Rarely it temporarily retains a certain amount of perception. In slowly developing detachments the visual disturbance is generally confined to the detached part, while in sudden ones the remaining portions often suffer also.

Central vision suffers most from detachments in the macular region, yet a reduced central vision may be observed when the macula is not directly attacked. This may be the result of a retinitic process or of a very flat, invisible macular detachment.

The light-sense almost always appears greatly reduced. In consequence such patients see much less when the sky is clouded or at dusk and at night. The color-sense is often impaired. The patients confound blue and green, which Leber thinks is due to the yellow color of the subretinal fluid, making the physiological fault similar to the character of color-perception of normal eyes during exposure to lamplight in which the yellow rays prevail. Color-blindness is rarely observed. Metamorphopsia may result from the movements of the detached retinal portion and cause the objects to appear blurred, bent, or notched. This symptom is constant and annoying. Photopsiæ are often present, fiery flashes, darts, and sprays of different degrees of intensity all being produced by the dragging on the retinal elements. Chromopsiæ are frequent, red, blue, and violet vision predominating.

When the detached part of the retina retains its perception the refraction is, of course, rendered hypermetropic, and may pass back to its original state if the retina returns to its normal position.

The macroscopical conditions in eyes with detachment of the retina were well known in preophthalmoscopic times. The exterior of the eyeball is usually unaltered, except in very old cases, in which at times a belt-like dimness (keratitis zonularis) is found in the cornea. The anterior chamber is generally very deep. The iris is often attached to the anterior lens-capsule. The posterior cortex of the lens, or sometimes the whole lens, becomes cataractous. In the vitreous humor, which in most cases is liquefied, different kinds of opacities will be found. When the detachment is small, the retina appears separated from the chorioid by a small quantity of coagulated fluid. When there is a larger partial detachment, this area appears like a trembling bag. In total detachment the retina forms a funnel, and is either firmly stretched between the optic disk and the ciliary portion of the retina or trembles. In the highest degrees the contracted, sometimes spiral, retina forms a cord or column running from the optic disk to the posterior pole of the lens. The ciliary portion of the retina is but rarely detached, because it is firmly united with the chorioid. I have described and pictured a very rare case of sudden detachment,[1] in which, after total exfoliation of the cornea through an abscess of this membrane following scarlet fever, and after prolapse of the lens and the largest part of the vitreous humor,

[1] Archiv für Augenheilkunde, Bd. xx. S. 102, Plate IV.

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the retina was detached, filled the perforation, and thus formed a retinal staphyloma.

In the detached retina hyperplastic processes which cause it to become thicker and to fill a large portion of the vitreous chamber frequently develop. Plates VIII. and IX., facing page 459, exhibit the condition in a case of this description. In rare instances the whole membrane appears otherwise normal, the chorioid being detached with the retina. I have reported such a case.[1] Plate VIII. shows also an extremely interesting condition of retinal detachment. Here the ciliary portion of the retina, with the uvea, is detached, while the remainder of the chorioid lies upon the sclerotic. In the deepest part of the retinal funnel a hyperplastic inflammation is started.

Sometimes spontaneous ruptures may be found in the detached retina. More rarely bone lamellæ, deposits of lime and cholesterin, or pigment accumulations in the detached retina may be seen. Equally rare are cysts, some of which become as large as a pea. Leber has reported and illustrated such a case.[2]

Microscopically, Nordenson has seen signs of a chronic inflammation in the chorioid with chorio-retinitic patches and chronic retinitis in the neighborhood of a spontaneous tear in three cases of myopic eyes with detachment. The shrunken vitreous humor consisted of wavy fibrillar bundles that adhered to the retina around the tear. At the same place some hæmatogenous pigment was found. Similar conditions were found in the three eyes. He also found fibrillar degeneration, shrinkage, and partial detachment of the vitreous humor in all the cases that he examined. Further, he determined the presence of partial adhesion between the vitreous humor and the inner surface of the detached retina. In all three of the cases there was a spontaneous rupture of the retina.

Aside from these conditions reported by Nordenson, signs of œdema or diffuse inflammation, hypertrophic degeneration, deformity and partial atrophy, with detachment of the layer of rods and cones, are sometimes found. Degenerative hyperplasia and atrophy of the nervous elements and the other layers of the retina, as well as cystic degeneration, sclerosis, and obliteration of the blood-vessels, all may take place.

In most cases retinal detachment develops rapidly, often suddenly. Frequently, however, it is preceded by prodromal symptoms, such as entoptic appearances, gray or black moving clouds like particles of soot, photopsiæ, like sparks dazzling before the eyes, and periodical dimness of vision. After such symptoms have existed for some time the detachment appears suddenly, being perceived by the patient as a dark cloud moving from one side over the field or as a dark curtain before the eye. At the same time objects near the cloud appear bent or broken. Visual acuity is at once more or less re-

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[1] Archiv für Augenheilkunde, Bd. xxi. S. 348, Tafel XIV. Fig. 7.

[2] Graefe und Saemisch, Handbuch der gesammten Augenheilkunde, Bd. v. S. 674.

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duced, even if the macular region is not implicated. The light-sense is impaired. The visual field shows a defect corresponding to the detachment. In very rare cases the affection may take a favorable course and terminate in a spontaneous cure.

The cases with the best prognosis are those of an inflammatory or a traumatic origin, as, for instance, in albuminuric retinitis. When the affection remains stationary for many years, it must be looked upon as one with comparatively favorable termination, especially if central vision is good. Such instances have been observed by von Graefe and others. In many cases the improvement is only temporary. Instances have been observed in which even improvement recurred several times before blindness finally came on. The most common course of the condition is very unfavorable. In the majority of instances the affection is incurable, and sooner or later leads to blindness. The detachment increases until it is total, and visual acuity and field are obliterated. During this process posterior synechiæ and posterior cortical and total cataracts frequently develop. These conditions are followed by the gradual reduction of intra-ocular tension. The eyeball gradually becomes smaller until total phthisis is developed. Even at this stage the patients may be annoyed by subjective light-perceptions and pain in consequence of calcification or ossification of chorioidal exudations. Possible development of sympathetic affection in the fellow-eye must also be thought of, especially in cases with posterior staphyloma of Scarpa.

Detachment of the retina is not a rare disease. According to Galezowski, who has seen it seven hundred and eighty-four times in one hundred and fifty-two thousand eye-patients, it occurs in about five-tenths of one per cent. of cases. In my clinical material from the years 1883 to 1893, among about sixty thousand patients I have seen only one hundred and forty cases,—that is, seventeen-hundredths of one per cent. In my practice as Provincial Oculist from 1863 to 1893, among about one hundred and twenty thousand patients I have seen two hundred and eighty cases of retinal detachment which could be ophthalmoscopically demonstrated,—that is, twenty-four-hundredths of one per cent. Men suffer almost twice as frequently as women, this being especially due to injuries and myopia. Detachments appear most often between the forty-fifth and sixtieth years; they are rare in youth, and in consequence of injuries become more frequent after the thirtieth year.

Bodily exertion, especially stooping and lifting of heavy loads, forced marches, hard riding on horseback or on the bicycle (I have seen such a case), coughing, sneezing, vomiting, pregnancy and parturition, anger, terror, venereal and alcoholic excesses, congestion, warm baths and steam baths, may all be mentioned as among the most prominent mediate causes.

Retinal detachments may be divided into the following etiological groups:

Primary Traumatic Retinal Detachment.—Widely differing types of injuries lead to immediate or less rapid detachment, especially contusions

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or wounds of the hard membranes of the eyeball, in which loss of vitreous humor and hemorrhages play an important part. The condition may also be produced by the lodgement of foreign bodies in the eye and by operations such as simple extraction of cataract, accompanied by loss of vitreous humor, especially when intra-ocular tension is increased and the walls of the blood-vessels, principally in the chorioid, are degenerated. Further, operations for staphyloma and iridectomy for hemorrhagic or malignant glaucoma, and non-traumatic cases in which, after extensive perforation of the cornea, the lens and a part of the vitreous humor prolapse, may all produce the condition.

Secondary Traumatic Retinal Detachment.—Such a condition is produced when injuries induce purulent or plastic inflammations in the chorioid, or when cicatricial shrinkage during the healing of a scleral wound or chorioidal rupture takes place. Small foreign bodies which become encapsulated, or synechiæ and consecutive shrinkage of the vitreous humor after a variety of operations, may produce the condition.

Retinal Detachment of Myopia.—Most cases of detachment appear in eyes in which there are high degrees of myopia. In low degrees of this refractive error the condition is rare. In most cases posterior scleral staphyloma is observed. Myopes of lower degrees with detachment are usually old people. In most cases the detachment is preceded for a prolonged period by changes in the interior of the eye, as liquefaction, opacities and detachment of the vitreous humor, and chorioidal changes in the macular region. From its etiology myopic detachment is very unfavorable, because its arrest or spontaneous healing is seen only in the rarest instances, and because the second eye is also generally attacked.

I have had occasion to observe a case of myopia which shows how much the retina may be stretched without being detached. It was one of the axial type of the highest degree, which I consider unique, since I have not found its counterpart in all literature; the length of the optic axis being forty-three millimetres. The case was that of a teacher, who in 1865 was seventy years old. He was very small, had a slight kyphosis and extremely prominent eyes. The left eye, which was still of some use, was barely covered when the lids were closed. The punctum remotum of this eye was at about twenty-six millimetres. The cornea appeared hemispherical, the anterior chamber was deep, and the iris showed some traces of trembling. There were floating opacities in the vitreous humor. There was no circumscribed staphyloma of Scarpa, but the whole posterior pole of the eye formed a diffuse posterior scleral staphyloma. In the macular region the chorioid was atrophic. The right eye was still more prominent than the left. Its cornea was almost wholly cicatricial, and contained new-formed blood-vessels. The fundus could not be seen. Vision was reduced to light-perception. This eye had been blind for years in consequence of a purulent process in the cornea which led to perforation. Continually recurring inflammations of the cornea induced the patient to have the right

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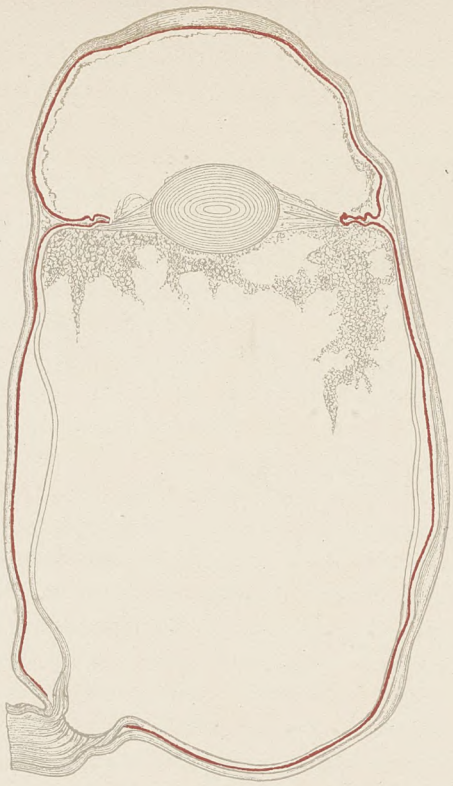
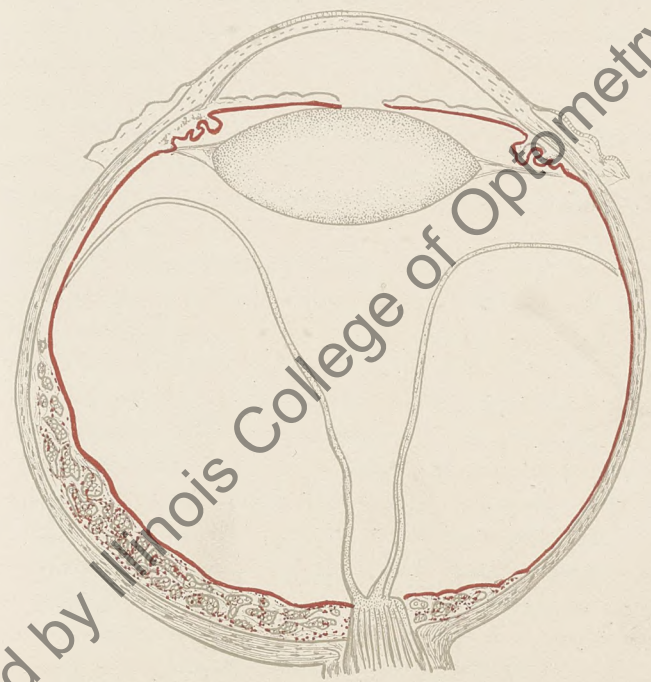
PLATE XVII.



Section of eyeball showing retinal detachment in myopia.

PLATE XVIII.



Retinal detachment from chorioidal carcinoma.

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eye removed. The cornea was almost totally replaced by scar-tissue, in which there were numerous blood-vessels and round-cell infiltrations; its posterior surface was covered with uveal pigment coming from the iris. The anterior chamber was very deep and contained some fibrinous deposits. The lens was in its normal position, the vitreous humor behind it containing fibrillar opacities. The retina was nowhere detached, though in places it was very thin, as also was the chorioid, especially in the macular region. The optic disk was markedly displaced inwardly. Plate XVII. gives an accurate picture of the condition.

Retinal Detachment from Irido-Cyclitis.—Irido-cyclitis but rarely causes a detachment of the retina which is visible with the ophthalmoscope. It is due to slow inflammatory processes that produce exudation into the anterior part of the vitreous humor, which shrinks and produces retinal detachment. When to this a hyperplastic retinitis is added, the picture of an amaurotic cat's eye may result and the detachment may appear as a so-called pseudo-glioma. Anatomically cyclitic detachments are found in blind eyes from progressive bulbar atrophy or phthisis bulbi, in which a clinical examination of the interior of the eye has been impossible. Such cases have been described by Pagenstecher, Noyes, Schiess-Gemuseus, and others.

Detachment of the Retina from Chorioiditis.—This form of detachment is practically the same as the cyclitic variety. Cases in which the diagnosis can be made with the ophthalmoscope, and the chorioidal primary affection diagnosed, are extremely rare. It usually consists in a slow degenerative chorioiditis, which, furnishing but scant inflammatory products, leads, however, to obliteration of the blood-vessels and atrophy of the stroma of the chorioid, which in turn induces detachment of the retina with hyperplastic inflammation. Such a case has been described by Haab[1] as a pseudo-glioma. Disseminate chorioiditis with detachment has sometimes been found, but it is difficult to decide whether this condition was a cause, a consequence, or a mere accidental coincidence. Cases in which the chorioidal affection cannot be clinically diagnosed are included among the idiopathic detachments.

Retinal Detachment from Retinitis.—In rare instances detachment due to different forms of retinitis, especially the albuminuric variety, is found. This may also happen in syphilitic retinitis, papillo-retinitis, and hyperplastic retinitis. In many of the rare cases of retinitic detachment it seems as if a complicating chorioiditis produced the detachment.

Retinal Detachment from Hemorrhages.—Detachments due to subretinal hemorrhages from the chorioid or retina appear ophthalmoscopically of a dark brownish-red to a black tint. Such spontaneous hemorrhages are seen in young people, anæmic youths or chlorotic girls, particularly among those with a hemorrhagic diathesis. It is found particularly during the summer months. Old people placed under conditions in which retinal

[1] Archiv für Augenheilkunde, Bd. xxiv.

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hemorrhages or hemorrhagic retinitis, especially from atheromatous degeneration of the blood-vessel walls, from heart-lesion, etc., may take place, are peculiarly prone to it.

Retinal Detachment from Purulent Changes.—Chorioiditis, retinitis, or chronic purulent chorio-retinitis may all lead to smaller or larger subretinal accumulations of pus which produce detachment and may give the picture of amaurotic cat's eye, which, as so-called pseudo-glioma, can only with great difficulty be clinically distinguished from real glioma. Such cases are observed after cerebro-spinal meningitis in children, after acute exanthemata, and sometimes after injuries, especially after perforation into the interior of the eye of pieces of caps, etc. In rare cases it seems to take place spontaneously.

Retinal Detachment from Cystic Degeneration.—Cystic formations may, when the cyst-wall which is situated towards the chorioid disappears from atrophy, give rise to retinal detachment. In general it is impossible to differentiate ophthalmoscopically between a large retinal cyst and a partial detachment, except, perhaps, by means of the absence of motion. The condition is found mostly during anatomical examination.

Idiopathic Retinal Detachment.—There is a large number of detachments the etiological factors of which are unknown. They are sometimes observed to develop slowly or suddenly in people of advanced age, and occur either spontaneously or secondarily, as, for example, after congestive conditions. Sometimes liquefaction of the vitreous humor may have been previously seen. These cases are more frequent in youth and in old age: in the one case the condition is due to a slow chorioiditis, and in the other to senile degeneration of the chorioid.

Congenital Retinal Detachment.—Walter has described as such[1] two cases which were seen in the clinic in Zurich. In one of these a coloboma of the optic nerve sheaths and macula, in the other telangiectatic chorioidal blood-vessels, were considered as the causes.

Retinal Detachment from Neoplastic Formations.—Sarcomata of the chorioid not only detach the retina in proportion to their growth, but lead early to a much more extensive or even a total retinal detachment, which may render the diagnosis of the primary affection difficult. The belief that the vascular neoplasm can secrete a subretinal fluid, or that it may, by pressing upon the chorioidal vessels, lead to a considerable stasis-hyperæmia, has given rise to the opinion that no detachment can be observed, the tumor soon perforating the retina and growing into the vitreous space.

Gliomata of the retina act similarly, especially the form known as glioma exophytum. The variety spoken of as glioma endophytum usually does not cause detachment. Less is known concerning detachment from the rare metastatic carcinomata of the chorioid. I have seen such a case in a forty-six-year-old woman who consulted me on account of decreasing

[1] Inaugural Dissertation, 1884.

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vision in the left eye. Two years previously a carcinomatous breast had been removed, followed in a few weeks by a local relapse with induration of the axillary and the supra-clavicular lymph-glands. The right eye was normal. Exteriorly the left eye appeared undisturbed. The ophthalmoscope revealed clear media, but to the temporal side of the optic nerve head an oval patch with a longitudinal diameter of three disks' width, with a somewhat smaller transverse one, could be seen by the inverted image. Its color was dirty yellow, with a few dark pigment-spots, thus contrasting it conspicuously with the remainder of the fundus. In the erect image it was found that the retina covering the tumor protruded for from three to four diopters' height into the vitreous humor. Small blood-vessels could be doubtfully seen shining through the centre of the mass, which from their ramification were evidently not retinal in character. The perimeter showed a corresponding defect that came close to the point of fixation. Central vision, which was unimproved by lenses, had fallen to one-twentieth of normal ($\frac{6}{120}$). I unhesitatingly diagnosed metastatic chorioidal carcinoma.

Two weeks later the patient returned, blind. Floating opacities could be seen in the vitreous humor. The retina was detached downward and towards the temple and hid the tumor. After another two weeks the patient reported, complaining of an unbearable pain situated in one-half of the head and in the eye. Intra-ocular tension was increased to plus two. The retinal detachment was total. On account of the pain, enucleation was proposed. This was gladly acceded to, and was followed by instant relief. Two months later the patient died from the original disease. One of the sections through the whole eyeball is pictured on Plate XVIII.

A second case which I observed was unique. This was one of a retinal detachment that developed slowly, almost concentrically, and in a comparatively brief period of time, in an eye with precorneal pigmented carcinoma. Total blindness soon appeared. When the patient—a thirty-one-year-old hotel-keeper—consulted me in 1883, the corneal tumor was visible on the right eye. The man, a stupid subject, was strongly built, had never been sick, and had come from healthy parents. He stated that years previously he had been struck on the right temple with a beer-glass. There was no scar. He attributed his affection to this injury, and thought that since the accident a nodule had formed on the eye. No other history could be got from him. Long after operating upon him, he told me that he had twice previously consulted me. I ascertained that I had seen him in 1881 and had then found a wart-like elevation on the cornea, the nature of which was not clear. Another entry in my case-book, made in May, 1882, showed that at the time of this visit there was a brown tumor, of the size of a hemp-seed and of soft consistency, situated on the temporal side of the cornea. Central vision in this eye was greatly impaired. The ophthalmoscope revealed a ring-shaped circumpapillary detachment which on the temporal side reached close to the macula. This detachment was most

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developed downward, and surrounded the optic nerve head like a wall. At that time I considered the case as one of malignant tumor, and noted it as being a probable precorneal pigmented sarcoma, proposing its removal.

At the time of the reappearance of the patient in 1883 the tumor was found to be the size of a pea, and was yellowish brown in color and soft. Intra-ocular tension was reduced. The anterior chamber was deeper than normal, and the pupil was somewhat larger than that of the fellow-eye. The iris reacted sluggishly to light. A gray reflex deeply situated behind the pupil could be indistinctly seen with the naked eye. There was a total, funnel-shaped detachment of the retina. Vision was reduced to light-perception. In spite of this the patient was not conscious of being blind in the affected eye, and was greatly astonished when I demonstrated it to him. At that time my diagnosis was precorneal pigmented sarcoma with little pigment (since some parts were brown, others yellow, and others almost white, and the consistency seemed to be that of a sarcoma), with total detachment of the retina. I recommended enucleation, to which he acceded after some reluctance.

A good section of the eyeball is shown in Plate XIX., while the appearance of the tumor can be seen in Plate XX. The histological diagnosis of the tumor was not sarcoma, but pigmented carcinoma, which surely was not to have been expected, in view of the patient's youth, the soft consistency of the growth, and its pigmentation. Its slow development and its poor vascular supply alone might have pointed towards a correct clinical diagnosis. The whole mass had a connective-tissue stroma, in which blood-vessels were embedded. In the interstices of the stroma lay nests of epithelial cells, which in places were pigmented.

What makes this case unique is that it was a purely precorneal growth, originating solely in the cornea and covered all over by Bowman's layer and the epithelium, while all other cases of so-called precorneal tumors thus far reported have started from the limbus and have gradually grown over the cornea. The form of detachment accompanying this growth also was unique, as it does not seem possible to connect it with the injury received years previously, since when he was first seen in 1881 his vision was intact.

Retinal Detachment from Subretinal Cysticercus.—Subretinal cysticercus by its growth as well as by its movements produces processes of irritation which soon lead to detachment of the retina. This detachment, although soon opaque, usually allows a shining reflex to be seen from the place where the cysticercus rests. Moreover, membranous opacities, with an almost characteristic system of folds and stripes that tremble when the eye moves, develop in the vitreous humor. This symptom-complex renders the diagnosis of subretinal cysticercus almost certain for the experienced observer.

Although high grades of retinal detachment can be diagnosed with the naked eye, and although they have been so determined during preophthalmoscopic times, it is usually necessary to make a careful ophthalmoscopic

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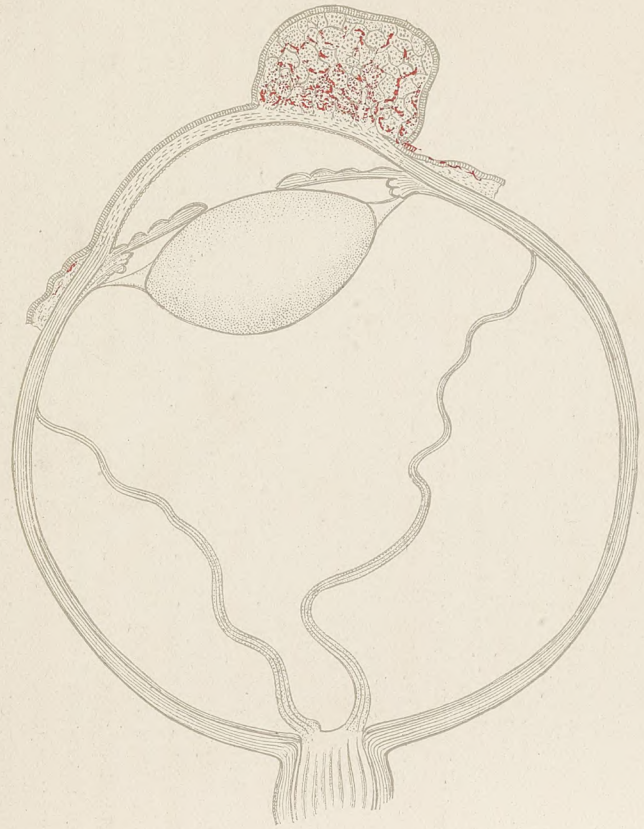
PLATE XIX.



Section of eyeball showing retinal detachment with precorneal pigmented carcinoma.

PLATE XX.

Section of cornea showing precorneal pigmented carcinoma.

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examination in order to be certain of the diagnosis, since externally noticeable symptoms, such as reduced tension, deep anterior chamber, dilatation of the pupil, and sluggishness of the iris, are not sufficient signs for a definite opinion.

When examining the vitreous humor of an eye with detachment of the retina, floating opacities are generally found. In a certain direction sudden discolorations of the fundus can be recognized, the discolored area appearing of a dull red or gray, gray-green, or bluish-white color. In this portion of the fundus the characteristic ramifications of the dark-red or blackish retinal vessels can be recognized by careful focussing. Trembling motions of this discolored part of the retina and of the bent and interrupted blood-vessels upon it can also be observed. The difference between the refraction of this place and that of the normal portions of the fundus is especially important in making a diagnosis. When the eye is emmetropic, the detached part becomes hypermetropic. If the eye be myopic, the detached area may become less myopic, emmetropic, or even slightly hypermetropic. If the eye be hypermetropic, the detached area will become more so. When there is a tear in the detached portion, the chorioid can be seen through it. When the lens is cataractous, the impaired light-sense, the disturbed projection, and the softness of the eyeball are all useful guides as to the diagnosis of the condition.

The prognosis of retinal detachment is generally very bad, permanent restitution of vision being obtained in the rarest of instances. The most favorable cases are those which develop with albuminuric retinitis, during pregnancy, inflammatory affections of the chorioid, disturbed circulation, diseases of the orbit, and traumatism. Prognosis is bad when the detachment is dependent upon myopia and in idiopathic cases. Partial detachments may remain stationary for a long time, or may change their locality by sinking deeper and thus give rise to a passing improvement. Any hoped-for favorable result is made still more uncertain by the fact that most patients wait a long time before they seek help, because the disease often develops slowly and causes no pain.

As may be seen from the foregoing remarks, the treatment of detachment of the retina is one of the most thankless labors of the physician. It is therefore not astonishing that in the course of time many different remedies have been used and the most diverse surgical measures tried. Formerly the entire antiphlogistic apparatus was brought to bear on this disease; and it is done sometimes to this day. Venesection, cupping, setons, sinapisms, vesicants, irritating foot-baths, depletion by means of leeches or the artificial leech of Heurteloup, have all been tried. Of the drugs, mercury and the iodides have been used both externally and internally. To these have been added diaphoretic measures by means of teas, wood extracts, jaborandi, salicylate of sodium, and injections of pilocarpine. Placing the patients in dark rooms, with rest on the back and compress bandages, has also been urged. Unfortunately, the results thus

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gained have been disproportionate to those of the tedious, painful, and even dangerous treatments employed, so that Galezowski, in 1887, stated that by these means he had seen cured but seven out of seven hundred and eighty-nine cases, and that of these only a couple had regained function, while in the others the retina had simply become readapted.

In 1859 Sichel proposed surgical interference. He introduced the plan of tapping the subretinal fluid by a puncture in the sclerotic and chorioid. In 1863 von Graefe recommended discission of the retina, and up to 1866 had employed it fifty times. By this means he obtained improvements, but they were not lasting. Bowman, Arlt, Secondi, de Wecker, and others accepted this method, but had no better results. The method used was that of introducing a needle from in front, the plan being based on the favorable effect of spontaneous ruptures of the retina. In accordance with Grizon's (Paris, 1877) communication, de Wecker tried by means of his "anse à filtration" to pass a fine gold wire through the sclerotic and chorioid, so as to allow the continually collecting subretinal fluid to be drained and thus to help the readaptation of the retina. This method not only proved unsuccessful, but was even dangerous, producing sympathetic disease in the fellow-eye.

In 1890 Galezowski advocated the plan of entering a catgut under the retina and stitching the retina to the chorioid, as well as his earlier recommendation to heal the retina into a scleral wound. No conscientious and experienced ophthalmologist could follow such counsel. The same author's plan of aspiration of the subretinal fluid with a Pravaz syringe has found very few followers. Later de Wecker modified von Graefe's method of discission and combined it with Sichel's plan of puncture, by discission of the retina from behind after a previous puncture. The operation, however, was devoid of good results, and sometimes was followed by very bad ones, so that he has acknowledged the danger of this method. Dislaceration with two needles was advised by Bowman, and was performed in a manner similar to de Wecker's operation for secondary cataract. The results were meagre and valueless. In 1872 iridectomy was proposed by Galezowski. It was then highly recommended by Castaldi, and often tried by Westhoff, Bettremieux, Gross, and others. Its successes, however, did not encourage imitation. From the above it is clear that all these operative measures have yielded few good results, and cannot be used to make the prognosis more favorable.

In 1872 Galezowski proposed the method of injections of tincture of iodine into the subretinal space. Abadie saw one case of cure after such an injection. Several times, however, he had bad results, causing him to employ Lugol's solution instead, after having first allowed the subretinal fluid to escape. In 1889 Dubarry found no favorable results, while Gelpke during the same year reported one death from meningitis following an injection of iodine, made under antiseptic precautions. During 1889 and 1890 Schöler materially modified this method, adapting it to the Leber-

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Nordenson theory, and employed it assiduously. He made injections into the preretinal space, in order to tear the fibres in the vitreous humor and to produce an adhesive retinitis which might counteract the traction of the shrinking vitreous mass and favorably influence the absorption of the subretinal fluid. From experiments on animals he considered the tincture of iodine to be the best remedy. He had a special instrument made, consisting of a Pravaz syringe with a small knife for the cutting of the vitreous bands. At the same time he injected a solution of twenty-five-hundredths of biniodide of mercury and two and fifty-hundredths of iodide of sodium in twenty-five parts of distilled water into the muscles of the back, preceding its injection by an injection of morphine. He praises the favorable effect of this preparation of mercury. The patients thus operated on were bandaged for a week and kept on their backs while mydriasis was maintained. In 1892 Schönfeld gave a *résumé* of the cases operated on after Schöler's method in the clinics of Pflüger, Maklakoff, Cofler, Dufour, Mooren, and Schweigger. In these statistics there were nearly one hundred cases, with twenty-three immediate successes, though, of course, the time of observation was too brief to allow of any conclusions as to the value of the results.

In 1894 Gillet de Grandmont described the cure of a myopic detachment by electrolysis, though de Wecker and Masselon two years previously had recommended galvano-puncture. In 1895 Deutschmann proposed two methods, both based on the Leber-Nordenson theory. The first was to cut the vitreous bands extensively and let the subretinal fluid escape. To this end he entered subconjunctivally a double-edged linear knife through the sclera, chorioid, and retina into the vitreous space, and pushed it to the opposite side of the eyeball, withdrawing it carefully, and cutting to both sides. His aim was to evacuate the subretinal fluid, to cut the retina in two places, to incise the vitreous bands, to release any preretinal fluid, and to free the retina so that it could be pressed against the chorioid by the preretinal fluid. In 1895 Jaencke reported ten cases operated on by this method, and stated that in the majority the retina was readapted. Here, as before, however, the time of observation was too brief for any definite judgment. Deutschmann himself warns against too enthusiastic hopes. His second measure, that of tapping the subretinal fluid and injecting the vitreous humor of the rabbit, is not necessary to discuss.

From all this it would appear that operative measures promise little help in detachment of the retina. Probably the best that can be done in high-grade progressive myopia is to enjoin the greatest care of the eyes and the avoidance of any congestion to the head, and to have the patient lead a temperate life.

In quite recent cases in strong people local depletion, rest on the back, and a compress bandage for a couple of weeks may be employed. If there is no change after this time, the case is hopeless. Diaphoresis by means of injections of pilocarpine, with Samelsohn's compress bandage and

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rest on the back, is sometimes serviceable. To this might be added the making of one or more punctures after von Graefe's method. With regard to the more important operative measures, further experience must be obtained before judgment can be passed on them.

GLIOMA OF THE RETINA (GLIOMA RETINÆ).

Glioma of the retina is a malignant intra-ocular tumor found in infancy, which starts from the retina and after a period of intra-ocular growth leads through increased pressure to ectasia (less frequently to irido-cyclitic processes with temporary shrinkage of the eyeball), piercing the eyeball, with a constant tendency to local relapses and metastases, until it finally kills the patient in from two to three years' time.

This formulation of what we call glioma, however, dates back only to 1853, when Virchow inaugurated the doctrine of the neuroglia. Although such a cementing substance had been previously described by Reil, Villars, and Keuffl, it was thought to be a fibrous tissue. Virchow's glia, a semi-fluid, granular cementing substance with nuclei, was opposed by Henle, Bruch, and others, but was confirmed by Bidder, His, and other observers; while Simon, Fleischl, Golgi, Gerlach, and Klebs have all declared it to be genetically identical with the nervous elements, differing in function only. At present Virchow's glia is recognized by probably all histologists. Neuroglia is found in the brain, in the spinal medulla, in the conarium, in the retina, in the acoustic nerve, and in the coccygeal and supra-renal glands. Diffuse hyperplasia of the neuroglia produces hypertrophy of the organ concerned, while circumscribed, tumor-like hyperplasia, according to Virchow, forms gliomata. Gliomata may be found wherever, in the normal condition, there is neuroglia.

There is no doubt that gliomata have occurred in the course of the past centuries and have been observed and treated. Yet the descriptions are so imperfect and the names are so confusing that it is impossible to state in which cases the condition as understood now was seen. Such cases are reported by Pavius, Fabricius Hildanus, Tulpius, Maase, and others, and by them were called fungus oculi, excrescentia fungosa, exophthalmia fungosa, tumor cancrosus, cancer, etc. The case seen by Bartholinus in 1665 may be concluded with moderate certainty to have been one of glioma. He advised the removal of the growth with the eye, "ne per nervos vitium ad cerebrum perveniret."

With the beginning of this century examples have appeared in literature which can with more certainty be looked upon as gliomata, and it is the merit of Hirschberg to have sifted them. He had the rare opportunity to examine specimens of the classical growths of old times, preserved by Johannes Müller in the Berlin Museum; besides, he had a large material from von Graefe's clinic and his own practice. The results of his examinations he gathered together in his monograph, "Der Markschwamm der Netzhaut," 1869.

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The first good description of glioma was given in 1809, by Wardrop. It was based on seventeen cases observed under the name of fungus hæmatodes, the malignity of which he well knew. He also understood the nature of metastases in the skull, and even the origin of the growth in the retina. His teachings were accepted by Saunders, Astley Cooper, Panizza, Pockels, Mackenzie, Linke, and others. In 1800 Burns termed such tumors fungus medullaris. Abernethy named them medullary sarcomata, while Maunoir made a distinction between fungus medullaris and fungus hæmatodes, applying the latter name only to very vascular growths, and perhaps to what are now called sarcomata.

In 1819 Laennec introduced the name encephaloid, which was accepted by Sichel, Robin, and most French physicians. Hooper spoke of cephaloma, and Craige of encephaloma.

The correct conception of Wardrop and his successors concerning the origin of the glioma in the retina was later forgotten, and the most varied organs were named as its original seat. In modern times the works of Virchow, Hirschberg, von Graefe, and others have proved that the explanation given by Wardrop was the only correct one, and that intra-ocular gliomata always spring from the retina. As their special origin, the inner granular layer is usually mentioned. It seems, however, that it may arise from different layers of the retina. I have seen a glioma in its earliest stages starting from both granular layers.

The growth of microscopical science at first did little to clear up the nature of glioma. The observations of Langenbeck and Robin on the identity of the glioma elements with those of the granular layer gave rise to Sichel's incorrect teachings of pseudo-encephaloid, which brought confusion into our knowledge of glioma which partially continues to this day.

Glioma of the retina may develop under two forms,—namely, as circumscribed glioma and as diffuse glioma. The former, which is found in most cases and which grows from the retina as a tumor, may either extend forward into the vitreous humor (glioma endophytum) or pass backward into the subretinal space (glioma exophytum). Each may sooner or later produce detachment of the retina.

With glioma endophytum a detachment is hardly ever seen, and rarely is it found with diffuse glioma. With glioma exophytum it is usual and of high degree. Glioma may very early spread heteroplastically into the chorioid and the optic nerve, though this more rarely takes place by continuity than by dissemination. Regressive metamorphoses, too, may occur very early.

Concerning its anatomical growth, I distinguish three forms:

1. *Simple Glioma of the Retina.*—This name I give to those forms in which, as is usual, regressive metamorphoses occur very early, and which, starting in the nucleus or in the protoplasm or in both, produce nucleus-death in the cells. These cells, which swell and finally become necrotic, can no longer be stained by hæmatoxylin. It is generally the oldest parts of

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the growth which succumb to this necrobiosis, and in which the results of the regressive metamorphosis, such as fatty degeneration, calcification, caseous or colloid degeneration, and hæmatogenous pigmentation, are found. The blood-vessels of these parts degenerate, their walls grow thicker and exhibit a colloid-like degeneration, while death of their endothelium takes place. During this time more or less profuse hemorrhages occur. In spite of these degenerative processes, the tumor continues growing in other directions, gradually fills the eyeball, and finally may exophthalmically reach a very large size. In such tumors fresh glioma cells (stained by hæmatoxylin), mixed with cells in different stages of necrobiosis, are found. Between them patches of products of regressive metamorphosis of different kinds, extravasations and ichorous masses, can be seen, while the blood-vessels, which have thickened walls, are partly obliterated or partly thin and ectatic. I have also found diverticula and retia mirabilia.

2. *Glioma Retinæ Luxurians.*—This term I apply to a rarer group of gliomata in which, for a long period, regressive metamorphoses are not observed, or concern only very small portions of the growth. In such tumors all the cells remain fresh and alive and can be stained with hæmatoxylin. Their blood-vessels show hardly any signs of degeneration. The tumors usually preserve comparatively much longer the ramification of the blood-vessels that is characteristic of young gliomata. There are no large hemorrhages. These tumors grow relatively quickly, and in sections appear uniform from their original site to the edge of the exophthalmic mass. I have depicted such an exenterated, injected exophthalmic glioma retinæ luxurians on Plate XXI.

This twofold manner of anatomical growth has also been observed in other tumors, especially epithelial carcinoma or cancroid, which sometimes undergoes regressive metamorphosis and forms a so-called rodent ulcer, that ulcerates in parts and continues growing in the periphery (carcinoma epitheliale planum). In rarer cases of regressive epithelial carcinoma metamorphoses do not occur for a long time. They continue sprouting, appear fresh, and may even develop a condition known as pseudo-papillæ on their surface (carcinoma epitheliale luxurians or, perhaps, papilliferum). The same twofold manner of growth can be observed with tuberculous granulations. In most of these cases caseous degeneration with the formation of tubercular ulcers results. In other types no regressive metamorphosis takes place; they continue growing luxuriantly to a considerable size, are filled with new-formed blood-vessels, and sometimes form false papillæ on the surface. I have removed such a growth, which made an excellent specimen of such tubercular new formations, from the region of the lacrymal sac. I term these cases granuloma tuberculosum luxurians papilliferum.

3. *Crypto-Glioma of the Retina.*—In extremely rare instances gliomata may exhibit a different manner of growth. At first such cases grow after the first type, enter the vitreous chamber, and may fill the eyeball and be disseminated into the chorioid and the optic nerve. They then undergo

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regressive metamorphosis from a tendency of their own which may be favored by inflammatory processes in the eyeball, such as irido-cyclitis and chronic panophthalmitis. After the primary growth has thus degenerated, the fluid parts of the eyeball may be absorbed and the eye shrink in consequence of the inflammatory processes, so that it presents the picture of progressive bulbar atrophy or anterior or complete bulbar phthisis. After a varying interval the disseminated patches in the chorioid or optic nerve, or in both, begin to grow again. They then fill the eyeball, increase its size again, break through it, and lead to the well-known end. Such cases are called crypto-glioma.

The retina may be detached, there may be cysts in it, and processes of regressive metamorphosis may take place. The optic nerve is usually infiltrated with glioma cells; more rarely it becomes atrophic. The vitreous humor becomes disintegrated and disappears by absorption in proportion to the growth of the tumor. Cholesterin may be formed in it. The chorioid atrophies, or in rarer cases becomes inflamed. Purulent chorioiditis of the chronic or partial subacute types may be seen. The iris and the ciliary body are generally atrophied, as in genuine glaucoma. Sometimes plastic irido-cyclitis will be found. Changed osmosis produces endothelial hyperplasia on the anterior capsule of the lens, with formation of cataract. The lens may be dislocated into the anterior chamber and pressed out of existence. The anterior chamber gradually disappears. The sclera becomes thinned, staphylomatous bulgings are formed, or cirsophthalmos is developed. In the cornea, which is usually ectatic, inflammation which leads to suppuration may develop and thus make a chamber of exit for the neoplasm. In other cases the pressure brings about an atrophy of the membrane. Before the tumor escapes the eyeball becomes ectatic and is filled with glioma cells. There are then no traces left of the other parts of the eyeball except a few remnants of uveal pigment.

When the tumor has pierced the eyeball it spreads into the orbit, either by continuity or by dissemination, giving rise to exophthalmos, and even producing atrophy of the orbital walls. It may penetrate into the neighboring cavities. If it grows forward, it implicates the conjunctiva, which swells enormously. This is followed by ectropium, erosions, phlebectasia, etc., of the lids and neighborhood, with caries of the contiguous bones. Meningitis may result from perforation into the cranial cavity.

Glioma consists of small round cells, a scant intercellular substance, and many blood-vessels without a stroma of their own, which appear simply embedded in the intercellular substance. The round cells, which in hardened specimens are often polygonal and appear to have offshoots, have a size of from seven to nine micromillimetres. Their nuclei are from six to eight micromillimetres in size. This shows that there is little protoplasm, which may be lost during the preparation of the specimen, so that the glioma cells appear to be simply nuclei which are more or less like the granules in the retina. Large, ganglion-cell-like cells, as described by

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Da Gama Pinto and others, are of such rarity that they do not influence the histological character of the tumor. The same is the case with multinuclear and giant cells.

In the majority of cases the intercellular substance is extremely scant. Usually I have found it to be composed of a granular, fibrillar, or reticular structure. Yet I think that this is an artefact due to the hardening process, and that in fresh gliomata the intercellular substance is semifluid and homogeneous. The numerous blood-vessels which lie embedded in this substance are mostly thin. Later, however, their walls become thickened and obliteration results. I have found a characteristic ramification in young gliomata by means of my method of injecting enucleated eyeballs. They are all terminal vessels, like the retinal ones, and are in association with some cerebral arteries. The artery is always accompanied by its vein, and the finest arterioles form loops and join the finest venules. There is no anastomosing capillary net-work. In glioma luxurians this arrangement remains until the latest stages, and may be found even in the extruded portion. (See Plate XXI.)

In 1868 I succeeded in getting an excellent injection of such a form of recent glioma. It was in a child, three years old, whose father had accidentally noticed a white reflex. At the temporal edge of the optic disk I found a transverse oval tumor, the longest diameter of which I estimated to be seven and the shortest five millimetres. It rose steeply into the vitreous humor for about four millimetres' distance. Newly formed blood-vessels could be easily seen. Islets of products of a regressive metamorphosis could be also recognized. I at once pronounced the case to be one of glioma endophytum. I enucleated this eye and injected it with carmine-gelatin. A section of the tumor is shown in Plate XXII. In 1891 Perles described, from Hirschberg's clinic, a case of glioma of seven millimetres' diameter, accidentally found in a child that was under treatment for glioma of the fellow-eye. It was similar in many respects to an interesting case seen by Knapp. The case most like mine is one reported by Hirschberg from von Graefe's clinic in 1867; Jodke (Skokalski) and Schweigger have reported similar instances.

Since Virchow's teachings concerning glioma, almost all modern investigators, except Klebs, have accepted the fact that the gliomata start from the supporting fibres of the retina. Hirschberg and others have named the inner granular layer as the one in which it is formed. In this layer Da Gama Pinto found that the nuclei were mostly in process of division with karyokinetic figures. Yet it could not be denied that the external granular and the intergranular layers were also taking part in the process. Manfredi and Iwanoff considered the nerve-fibre layer to be the starting-point. It seems, therefore, possible for gliomata to start from all the layers of the retina which have supporting fibres,—that is, all except the rods and cones,—the inner granular one being, however, their most frequent starting-point.

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PLATE XXI.

Section of glioma retinæ luxurians.

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Section of glioma endophytum.

Klebs,[1] who thinks that there is no genetic but only a functional difference between glia and nervous elements, considers gliomata to be hypertrophies of all parts of the retina, and terms them neuro-glioma.

That glioma is a form of tumor that is independent of and different from sarcoma was first doubted by Cornil and Ranvier. Later it was termed small-celled retinal sarcoma by Delafield. Nagel agreed in this opinion. Even in one of the most recent papers by Becker we find this opinion reiterated.

From Virchow's stand-point the supporting tissue of the retina cannot be considered identical with common connective tissue; and from the stand-point of Klebs the gliomata are derived even from a different germinal layer than that from which the sarcomata arise. The clinical picture of glioma also is so well defined that to confound it with sarcoma is against good judgment. The reasons adduced by Becker to prove that gliomata are sarcomata are of an extremely doubtful nature.

Becker thinks that there is a sufficient number of mesoderm cells in the blood-cells to give rise to sarcomata. To this it must be answered that their origin from the granules of the granular layer directly, especially of the external one, can be observed, and that processes of division can be seen there, as Da Gama Pinto and I and others have witnessed, and that not a single observer has found a glioma starting from a retinal blood-vessel. With sarcomata this is nothing rare. The second reason of Becker's belief is still less tenable. This, in fact, is Eisenlohr's theory,—namely, that in embryonic life mesoderm cells of the vitreous humor reach the central retinal artery along the persisting branches of the hyaloid artery in communication with it, and in this situation cause a glioma to form. This hypothesis is a mere fancy, without the least observation to base it on, and hence does not call for serious consideration.

The most frequent mixed tumor of this kind is glio-sarcoma, as Virchow has named it. When spreading into the chorioid and sclera and exophthalmically, gliomata may sometimes change their character in that their cells grow larger, up to fifteen micromillimetres and more, while their nuclei remain comparatively small (ten micromillimetres),—that is, the protoplasm is increased, or, in other words, the cells assume the character of those of a small round-cell sarcomatous variety. Berthold was the first who asserted, as I think correctly, that gliomata may assume a sarcomatous character after having spread into the chorioid, and that this membrane, irritated by the entering of the glioma cells, furnishes the sarcomatous elements. In a reverse way it may occur that sarcomata of the chorioid take on a gliomatous character. This perhaps explains von Graefe's two observations of glioma in adults.

I have enlarged upon Berthold's theory in two publications,[2] and have

[1] Beiträge zur Geschwulstlehre.

[2] Max Schultze's Archiv für Augenheilkunde, Bd. xxv., and Rozprawy Ceske Akademii, ii.

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given it a firmer basis by observations upon an interesting and complicated mixed tumor which will be referred to later. To this group probably belongs the case of neurocellular sarcoma of the retina by Taillais and one by Cros, in which ossification was found. True sarcomata of the retina are of a secondary nature, coming from the chorioid and propagated into the retina. Sarcomata of the optic nerve and retina as observed by Schiess-Gemuseus are of metastatic character.

Fibro-gliomata are rarely observed. They are of two kinds. In the first, known as real fibro-glioma, the intercellular substance is better developed and is of a fibrous nature. Such a case, although not a typical one, has been described by Hulke, Waldeyer, and Kono. The second form, which I should call glio-fibroma, and which I have seen several times, results from the entrance of the glioma mass into the sclerotic. As soon as the glioma cells wander into the sclerotic its fibres are in places pressed aside like a fan, resulting in their hyperplasia. Thus a tumor in which the scleral fibres form a connective-tissue net-work, in the interstices of which the glioma cells are embedded, is obtained. Such a case is seen in Plate XXIII.

Telangiectatic glioma has been reported by Virchow. Da Gama Pinto has seen a glio-angio-sarcoma. To these types probably belong, as Leber also thinks, the case of Stendener, which was described as an alveolar sarcoma of the retina, and, finally, the case recently reported by Becker under the title of tubular angio-sarcoma of the retina. I have described and pictured a cylindro-glioma of the retina. Myxoglioma (of the sheaths of the optic nerve), as Goldzieher has described it, has, so far as I am aware, never been seen in the retina. The tubercular glioma of Da Gama Pinto is probably a mistaken observation.

The interesting mixed tumor which I mentioned above in connection with Berthold's theory is the following. In November, 1884, a boy was brought to me with an enormous tumor protruding from the right orbit. The father traced the affection to an injury. He had seen the shining reflex from the eye, but sought help only when a red mass of the size of an apple, and which bled easily, had grown out of the eye. At the hospital to which he had been taken the boy remained for four weeks, when the physician in charge declared the tumor to be inoperable. When I saw it the growth was as large as an orange. The boy was then taken home and treated with domestic remedies, while the tumor kept growing larger. The patient again came to me. He was pale and thin, but was otherwise healthy. The left eye appeared normal. From the right orbit a mushroom-like tumor half the size of a child's head protruded. Its color was pale red. Here and there it was livid, with yellow patches, and there were hemorrhages from the new-formed vessels, some of which were as thick as a quill. In places it was soft, like brain-substance, in others it was hard, while in some positions it was almost fluctuating. Its surface was tuberous, and was partly covered with a purulent ichorous mass and dry crusts. Its

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PLATE XXIII.

Section through a glio-fibro-sarco-myxo-angioma.

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vertical diameter was fully twelve centimetres, while its horizontal diameter equalled ten centimetres. From the orbital margin its highest point protruded from seven to eight centimetres. My diagnosis was glioma, and my prognosis was bad. Three days later I removed the tumor, together with the orbital contents and eyelids. This was a difficult procedure. The soft mass was, unfortunately, spoiled by a colleague while my back was turned during the stanching of the hemorrhage. In spite of this I succeeded in injecting it, entering more than sixty canules into the new-formed blood-vessels and ligating or grasping several hundred of them with a forceps.

The microscope revealed that the remnant of the eyeball occupied almost the centre of the tumor. Nothing could be recognized but the sclerotic, which was split into fibres in its posterior part and through hyperplasia of its elements formed a fibrous net-work filled with glioma elements. (See Plate XXIII.) The anterior part of the eyeball was densely packed with glioma cells. Between it and the posterior portion, which was changed to a glio-fibromatous mass, there was a line of brown pigment, the remains of what was once the chorioid. The optic nerve could be barely seen in the tumor mass. The tumor around the remnant of the eyeball consisted partly of glioma cells and partly of round sarcoma cells. Near its periphery large sarcomatous spindle cells could be seen. Close to the surface, especially inward, massive layers of unstriped muscular tissue were found. In some places the blood-vessels were immensely developed, so that these localities seemed to consist of blood-vessels. Formations like retia mirabilia and ectatic vessels, some of which had diverticula, were also found. Where the tumor had been in connection with the skin of the nose, it was covered with a layer of epithelium and there were immense and well-formed papillæ. In other portions wedges of epithelial cells entered the sarcomatous tissue, producing the appearance of an epithelial carcinoma.

I may say that doubtless this tumor was primarily a pure retinal glioma which, when it spread into the sclera, produced proliferation with the formation of a glio-fibroma. When the glioma cells spread into the orbital tissue they started a proliferation in this locality, and sarcomatous (round and later spindle) cells were formed, and thus a glio-fibro-sarcoma resulted. The same process was repeated with the unstriped muscular tissue of the blood-vessels, and thus the tumor formed a glio-fibro-sarco-myo-angioma. Where the tumor was connected with the normal skin the epithelium proliferated and grew over a part of the tumor, forming papillæ, and in places cancroid offshoots entered the underlying tissue, and thus was produced the most complicated of all mixed tumors.

I believe this case to be an important proof of Berthold's theory, since it seems evident that as the tumor came in contact with the different tissues it induced in them severally a hyperplasia characteristic of their own structure.

The boy soon recovered from the operation and was discharged apparently cured, but he returned in January of 1885 with a marked relapse in

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the orbit. The neighboring cavities were filled with the tumor, and cerebral symptoms showed that propagation had taken place into the cranial cavity. He died in March of the same year. I could not obtain the whole head. The excised parts of the tumor showed, strange to say, that the relapse was purely gliomatous in type.

According to the age of the glioma, we may distinguish between different stages, each giving a special clinical picture.

The initial stage, which may also be termed the ophthalmoscopic one, since the diagnosis can be made only by means of the ophthalmoscope, is observed only in extremely rare cases. The reason for this is that the infantile patients do not give any subjective signs as to the positive scotoma due to the tumor. It is most likely to be recognized, however, in bilateral gliomata, when the disease in the fellow-eye is so far progressed as to bring the little patient to a physician. At that time a whitish, somewhat prominent, spot, sometimes surrounded by smaller ones, can be seen in the fundus. New-formed blood-vessels, which by their ramification are at once recognized as different from the retinal ones, are generally found. When the case has progressed a little further, yellow spots, the results of regressive metamorphosis, with small hemorrhages, may also be observed. The prominence of the tumor can be proved by the parallactic movements or by means of convex lenses. From the refractive power of the convex glass necessary the height may be found.

The second stage is that of the amaurotic cat's eye. In this the exterior of the eye still appears free from irritation, yet in a certain direction a pupil-reflex can be observed, followed later by a manifest white or whitish-red or whitish-yellow reflex, which Arlt has likened to the sheen of gold, Saunders to that of silver, Scarpa to that of steel, Knapp to that of metal, Schindler to that of mother-of-pearl, Schneider to that of topaz, and von Weller to that of ruby. The readiness with which this reflex can be seen is due to the fact that the fundus is forced more and more forward by the tumor, and the refraction of the eye is rendered hypermetropic to the highest degree. When the reflex is examined more closely with strong lenses, with oblique illumination, or even with the naked eye (since the dioptric apparatus of the eye itself acts like a convex lens), a whitish, whitish-yellow, or whitish-red tuberous mass growing into the vitreous space can be seen. Upon this mass new-formed blood-vessels, white patches of degeneration, and small hemorrhages may all be observed.

At this stage the anterior chamber is shallow, the pupil is somewhat dilated, and the iris is sluggish. Intra-ocular tension is generally normal, while vision is abolished or reduced to perception of light. In rare cases, however, vision may be partially preserved even to the later stages of the disease. Generally there is no pain. It is at this stage that most cases are brought to the physician, since the strange reflex can be observed even by the least intelligent of parents.

The third stage is that of glaucoma or irido-cyclitis. As the glioma

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grows farther into the vitreous chamber, intra-ocular tension gradually increases and the clinical picture of the glaucomatous stage results. A continually increasing pericorneal episcleral injection with a bluish tint may be seen, which in rare cases may produce chemosis. The cornea shows slight diffuse opacities, and its surface becomes dull, as if it had been scratched with a needle. The anterior chamber grows shallower, the aqueous humor becomes turbid, the iris, which is immobile, shows a dirty discoloration, while the pupil is wide. Sometimes the lens is rendered opaque, and if any vitreous material remains this is also opaque. Instead of the previous golden or reddish reflex an indistinct gray one will be seen, thus often rendering a direct view of the tumor impossible. Tension rises to plus two, though rarely it goes higher, and atypical pains, which may reach a high degree, appear. When parents have not observed the condition of the amaurotic cat's eye, the patients are brought for aid at this stage on account of a continued complaint of pain, an easily recognizable inflammatory appearance, and symptoms of ciliary irritation. The glaucomatous state, however, is not always as typical as here detailed, because the infantile sclerotic is less resistant and becomes stretched before tension can be materially increased. In rare instances the development of a plastic irido-cyclitis instead of a glaucomatous condition may be observed.

The fourth stage is that of ectasy followed by atrophy. After a variable period of the glaucomatous stage the increased tension produces ectasy of the eyeball, of which there are two forms. In the first the cornea becomes ectatic, resulting in megalocornea or keratoglobus, just as with embryonic glaucoma according to the theory of Horner and Muralt. To this is soon added an ectasia of the anterior parts of the sclerotic, the resulting picture being that of hydrophthalmos or buphthalmos. In the second form the ectasia is not so uniform. Ciliary, intercalary, or equatorial staphylomata are formed, and the cornea suffers only secondarily. The picture is that of cirsophthalmos. In the rare cases in which, instead of an increase of tension, irido-cyclitis develops, the eyeball may for a time shrink instead of becoming ectatic. In this condition the picture of progressive bulbar atrophy or anterior or total phthisis of the eyeball will be seen.

Perforation of the eyeball by the gliomatous mass constitutes the next stage. When the globe has been filled with gliomatous tissue and its membranes have mostly atrophied and disappeared, the space becomes too small for further growth of the mass, and it begins to perforate the eyeball and to grow exophthalmically in different ways. It may pass through the optic nerve. If so, it may escape in two ways: first, as occurs very early, as a rule, the tumor, either by continuity or by dissemination, passes along the optic nerve fibres themselves, and by this road breaks the sheaths and extends into the orbital tissue; second, as usually happens later, it grows through propagation of the gliomatous masses into the intra-vaginal space backward into the orbit. Both forms produce the early appearances of

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exophthalmos. It may pass through the sclerotic. The glioma may pierce this tunic by the formation of staphylomata, atrophy from pressure, and consequent rupture, or it may extend through heteroplastic propagation of the glioma into the sclerotic, with loosening of its fibres and hyperplasia, followed by development of a glio-fibroma, and finally by protrusion of the glioma through this net-work. If the mass pass through the cornea, it may penetrate in three ways: first, in a mechanical manner by pressure and atrophy after the development of megalocornea followed by rupture; second, through heteroplastic propagation, when the glioma cells penetrate into the cornea, split it, and grow through it in a way similar to that seen in the sclerotic; third, through a secondary keratitis, which first appears in an interstitial form, followed by sloughing that transforms the cornea into a purulent area, thus opening a way for the exit of the tumor. In addition, it may find egress from the globe in a forward direction in the ciliary region through the minute canals through which the perforating arteries and veins of the anterior ciliary vessels enter and pass out; or it may escape in the equatorial region, in the places through which the vorticose veins make their exit. Another way in which the growth may pass outwardly is that posteriorly through the minute channels by which the posterior ciliary vessels and the ciliary nerves enter the eyeball around the optic nerve.

The next stage is that in which the growth has escaped from the eyeball and grows most rapidly. As soon as the mass has pierced the eyeball it extends very rapidly, spreading heteroplastically from tissue to tissue with which it comes in contact. It may spread forward through the cornea or the episclera and form an anterior extra-bulbar mass, involving the conjunctiva, the eyelids, and their surroundings by continuity, generally reaching the size of a man's fist or that of half of an infant's head or more before death ends its progress.

If it has perforated the eyeball in the equatorial region, or if it has passed backward through the optic nerve or its neighborhood, it first fills the whole orbit, producing exophthalmos. It next produces atrophy of the orbital walls, and spreads into the neighboring cavities and often into the cerebral cavity, when, after the appearance of severe cerebral symptoms, death ensues.

A propagation of glioma from one eye to the other by continuity through the optic nerves and chiasm does not seem to happen. There is only one such case, reported by Hjort and Heyberg, in which the growth had extended from one eye to the optic disk of the other, and would probably have reached the retina of the second eye had death not intervened.

After removal of the glioma local relapses are common, their occurrence being the more likely the later the operation has been performed.

The metastases of glioma in distant organs are produced by the agency of the blood-vessels. In 1871 Bizzozero found glioma cells in the blood-

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vessels in a case in which there was metastasis in the liver. Similar reports have been given by Thalberg, Waldeyer, and Landesberg. Metastases are most frequently found in the parotid and submaxillary glands; then in the mediastinal, mesenteric, and retro-perineal glands, and in the bones of the skull, in the sternum, and in the ribs. Other forms of metastases have been seen,—for example, in the liver, by Knapp and Rusconi; by Heymann and Fiedler, in the ovaries and in the kidneys.

Every glioma not interfered with leads, without exception, to death of the patient. This usually takes place in from one and a half to two and a half years after the beginning of the new formation. Death results from general marasmus or septicæmia caused by the septic products of the neoplasm, or from loss of blood, cerebral disturbances, or some intercurrent disease.

Crypto-glioma of the retina is a term, not yet used in ophthalmology, which I introduced for the first time in a paper written in 1893 in explanation of some rare ocular neoplasms seen in Bohemia. For me crypto-glioma means the condition opposite to that of pseudo-glioma. Among the crypto-gliomata I include those cases in which there is a glioma present, but in which even an experienced diagnostician is unable, at least for a certain period of time, to make a correct diagnosis, and at times may be forced to make an improper one. The name crypto-glioma is as correct as pseudo-glioma. As a rule, every case of pseudo-glioma means a diagnostic mistake, and it is plain that in the course of time, as our knowledge increases, pseudo-gliomata will become rarer. Crypto-gliomata, however, are true gliomata which have not been, and as yet cannot be, recognized in the early stages, the latter course of the affection and the anatomical examination alone revealing their true nature. Therefore they are such gliomata as run their course under the picture of a disease of a different nature, and thus lead to an incorrect diagnosis.

It is a well-known fact, repeated in every text-book, and in most cases without any experience of the writers (being simply recopied), that gliomata, instead of assuming the glaucomatous and the ectatic stage, may lead to an irido-cyclitic one with temporary atrophy or phthisis. Such types, however, are extremely rare. Hirschberg has collected the older ones in his monograph. In 1887 Grolmann reported an interesting case from von Hippel's clinic, which appeared in a boy whose one eye had been enucleated for glioma without relapse when he was two years old. Four years later the fellow-eye showed the symptoms of amaurotic cat's eye, due to whitish-gray movable masses situated in the vitreous chamber, and reaching up to the posterior pole of the lens. Blood-vessels could not be seen. Vision $= 20/cc$. Glioma was thought of, but there were reasons against this diagnosis. The gray movable masses appeared like infiltrations into the vitreous humor. The lack of vascularization and the comparatively good vision argued against glioma. On the supposition of an inflammatory process, mercurial inunctions and calomel were ordered, with the result

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that the vitreous humors cleared and vision rose to 20/xxx. Finally external symptoms of inflammation developed, tension increased, hypopyon appeared, the eyeball became ectatic, and death ensued amidst severe general symptoms. An anatomical examination of the ocular tissues revealed the existence of glioma of the retina with metastases into the vitreous humor. The author justly concluded from this interesting case that in the first stages gliomata may shrink and partly disappear. His second conclusion, that this shrinkage is assisted by mercurials, is not, however, altogether proved. In a case which will be detailed later I have observed shrinkage of gliomatous masses in the anterior chamber, twice in succession, without the use of mercurials.

In 1891 Jung reported an interesting case seen in Leber's clinic. In a girl four years old, after an iridectomy had been performed, amaurotic cat's eye, besides hypopyon and nodules in the iris, was observed. The diagnosis was tubercular irido-chorioiditis, this being rendered tentative probably by the fact that the mother was markedly phthisical. Anatomical examination revealed the presence of a glioma of the retina, small gliomatous nodules being situated in the iris, with free glioma cells in the anterior chamber. Although no shrinkage took place, the case must be considered as one of typical crypto-glioma.

In 1893 I saw a three-year-old girl who had been apparently healthy until her parents noticed a strange reflex from the pupil of her left eye some time previously. I found the exterior of the eye of this child, who was apparently strong and healthy, normal. The pupil was somewhat dilated, and the iris was sluggish. From the fundus came a whitish reflex. With the ophthalmoscope I saw whitish-yellow and whitish-red nodular masses which sprang forward from the fundus through about a third of the vitreous space. I could not with certainty see new-formed blood-vessels, though I imagined that they shone through in the red parts of the tumor. Tension was slightly increased. Vision equalled the ability to recognize movements of the hand at one metre's distance. I diagnosed glioma retinæ endophytum, and proposed immediate enucleation. The parents refused, and, as I insisted, I was discharged. For treatment the child was put into the hands of a shoemaker, who had married a woman who had been for a time a servant in Professor Arlt's house and had carefully collected old prescriptions from the waste-paper basket and brought them as her marriage portion into the family. The shoemaker made good use of these, and on them founded an ophthalmic practice which, by reason of the gullibility of the general public, flourished so well that before long he was considered to be the highest ophthalmic authority in Prague, his clientèle including persons of nobility, prelates, and officials. When he was convicted of quackery, numerous carriages drove up to the jail and people in high positions used their influence in his favor. After this he continued to practise, without interference, until his death.

Since the child grew continually worse while being treated by the

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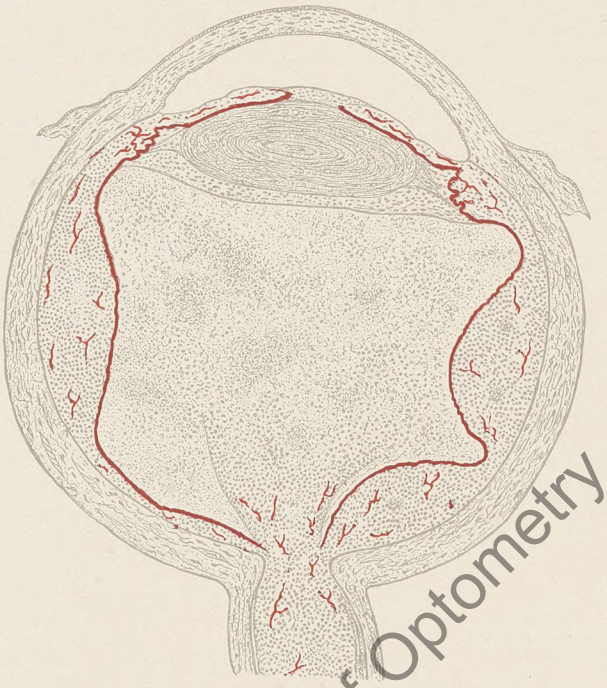
PLATE XXIV.



Section of eyeball showing crypto-glioma.

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shoemaker, I was again called to see her. I found the patient much altered. She was somewhat emaciated, pale, and fretful to the highest degree. There was so much photophobia that an accurate examination was rendered difficult. The eye showed some ciliary injection. It was smaller, and intra-ocular tension equalled minus one. Excessive pain could be produced by pressure on the ciliary region. The cornea was clear, the anterior chamber was normal, the iris was discolored and bound down to the lens-capsule, and the pupil, which was narrow, was filled with inflammatory products. The ciliary portion of the iris was somewhat retracted. There were pronounced idiopathic pains. The eye was blind.

The diagnosis was now clearly that of incipient progressive bulbar atrophy from plastic irido-chorioiditis, yet I did not allow this to sway me from my original diagnosis of glioma of the retina, being warned by the cases reported in literature which proved that with a true glioma such an irido-cyclitic stage and temporary shrinkage may occur. Furthermore, I not only maintained my former advice of enucleation, but urged it even more strongly, since the child's health was becoming more and more impaired and the blind eye was painful. The parents still refused, and demanded a consultation. I called my teacher, Professor von Hasner. He made the diagnosis of malignant plastic irido-cyclitis, and gave a bad prognosis. He ordered an energetic mercurial treatment, which was followed without success. The eye grew smaller, and tension fell to minus two. This lasted about two months. At the end of the second month I noticed a stand-still, and it even seemed to me later that tension rose somewhat. This became manifest, and tension rose to plus one and the eyeball grew larger. At the end of the third month the child contracted diphtheria and died, twenty-four hours after a tracheotomy had been performed.

I secured the eyeball. It was of extreme importance to me, because death had occurred just at the time when the process of shrinkage had changed into one of growth, and I could hope for important results concerning these processes. This hope was not deceived. I hardened the eye according to my own method and cut it into sections. (See Plate XXIV.) The globe appeared somewhat smaller. The sclerotic was thickened, and the cornea, with the exception of a few more round cells situated in its parenchyma, was normal. The anterior chamber was small. The iris was filled with round cells that were attached to the anterior lens-capsule. The pupillary area was filled with iritic products which consisted of round and spindle cells with a finely granular intercellular substance. Similar products filled the posterior chamber. Between the cortex and the nucleus of the lens there were a few cavities in which myelin-spheres and detritus lay. Behind the lens I found a large layer of cyclitic products consisting of round and spindle cells, intercellular substance, and a few new-formed blood-vessels. The vitreous space was filled with necrobiotic products which were composed of the regressive metamorphosis of glioma elements, that would not take any hæmatoxylin stain. The optic nerve was infil-

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trated with fresh glioma cells, that took the stain well, and from it a broad stream of fresh glioma cells, widening funnel-like, was found to extend into the vitreous space. The chorioid was considerably thickened throughout, and in places it was seen to protrude into the vitreous chamber, it being completely filled with fresh glioma cells. The unchanged pigment epithelium formed a well-defined barrier between the recent glioma of the chorioid and the regressive products of the old glioma in the vitreous space.

These highly interesting conditions I explain in the following manner. The primary tumor, which I had in the beginning correctly diagnosed as glioma retinæ endophytum, grew gradually into the vitreous space until it filled it, and at the same time it spread by dissemination into the optic nerve and chorioid. An acute irido-cyclitis, which was probably increased by the treatment of the shoemaker, was then produced. Under this treatment the whole tumor mass filling the vitreous chamber underwent a regressive metamorphosis, and the condition of progressive bulbar atrophy resulted. Later the disseminated germs began to grow in the optic nerve and chorioid, and thus the eye again grew in size and its tension became increased. I wish also to add that it would have been utterly impossible in the later stages of this case to diagnose glioma had I not originally made a careful examination. This is shown by the fact that so eminent a physician and so accurate a diagnostician as Professor von Hasner, in spite of my previous examination, could not be convinced that we had to deal with a glioma retinæ. Thus, for him at least, the case was in truth one of crypto-glioma.

Another case of crypto-glioma I saw in the years 1893 and 1894. A twenty-months-old infant was received into my clinic in November, 1893. The history showed that the father and the mother were healthy, and that the child had not passed through any severe illness. When the patient was two weeks old the mother had seen something in the pupil of the right eye which appeared like a yellow lentil, but from her description I could not determine whether this was a reflex from the interior of the eye or was a yellowish substance situated in the anterior chamber. About three months later inflammatory symptoms, photophobia, and lacrymation appeared. Apparently there was no pain. From this time, however, the eye began to increase in size until it reached the condition found when I first saw it. Two months before I saw her the parents noticed a little nodule in the anterior part of the eye (anterior chamber), which during the last two weeks had been growing rapidly. When I first examined the case the right eye was somewhat enlarged, so that it was just about covered by the lids, whose edges were red. Its shape was irregularly nodular, and in its front portion there were a few bag-like elevations. Its general color was a bluish-gray slate, yet other portions appeared yellowish brown and rose-tinted. The locality of what was the cornea would have been difficult to determine if it had not been for the iris-pigment that was situated on the posterior surface of the membrane. From the centre

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PLATE XXV.

External appearance of intra-ocular gliomatous formation.

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of this roundish portion an intensely white retracted scar, which was more than nine millimetres long and three millimetres wide in its broadest part, ran outward in rays. It was somewhat arch-shaped, and reached over into the inner ciliary region for about three millimetres' distance, where there was a bag-like elevation, which was reddish yellow in color, the size of a pea, and contained new-formed blood-vessels. The bulbar conjunctiva was hyperæmic. I could hardly decide whether the sensibility of the degenerated globe was increased or diminished, since the condition of the child and its continuous motions rendered an examination difficult. Tension was reduced to almost minus two.

The left eye, which appeared externally normal, showed a marked pericorneal episcleral injection. The cornea was normal. In the anterior chamber a whitish-red tumor that started from the lateral iris-angle, spread in a medial direction, and filled about two-thirds of the anterior chamber, could be seen. The tumor consisted of separate round nodules of the size of a swollen grain of farina, so that the mass looked like a piece of coarse farina gruel. The tumor was pervaded by new-formed blood-vessels which started from the lateral iris-angle and ran horizontally in a medial direction to the edge of the mass, and in one place extended downward, left the growth, and entered the iris. The iris was greenish in tint and its markings were dull. Upward the iris appeared to be wanting. A careful examination, however, revealed that in this position its pupillary margin was tilted backward, so that an apparent coloboma resulted. Through this coloboma between the upper edge of the tumor and the ciliary margin of the iris I could with difficulty look into the interior of the eyeball. I there saw a light-gray reflex, but failed to perceive any details of the fundus. Vision was reduced to perception of light, and intraocular tension had fallen to minus three.

During the next two weeks some changes took place. The right eye became slightly enlarged. The bag-like elevation in the inner ciliary region markedly increased, fluctuated under the finger, and protruded so greatly that a perforation in that position became imminent. The exterior of the left eye remained unchanged, but the tumor in the anterior chamber grew steadily by the renewed apposition of nodules at its medial edge, so that the free anterior chamber was increasingly reduced. At the same time small foci of regressive metamorphosis became visible in the lateral iris-angle in the position from which the tumor sprang. These foci appeared yellowish gray, like caseous detritus. A series of drawings illustrating these stages is shown in Plate XXV.

I concluded, from the nodular elevations, especially of the anterior part of the right eyeball, from the scar of the cornea and ciliary region and the iris-pigment shining through, from the bag-like elevation which by its fluctuation showed a renewed perforation to be imminent, and from the reduced tension, that, since no injury had occurred and the first pathological symptom had been observed twenty months previously as a yellow reflex, I had

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to deal with a spontaneous suppurative process of the uvea (a so-called chronic or subacute partial purulent chorioiditis), which had produced a sloughing of the cornea and a portion of the sclera in the outer ciliary region with perforation. From the fact that the old scar reached into the external ciliary region and that a new perforation was about to take place there, I, in addition, concluded that the morbific process had probably started from the inner portions of the ciliary body.

A true granuloma, which on the lateral side had entered the anterior chamber through Fontana's space and here had gradually spread medially, could be seen in the anterior chamber of the left eye. It appeared to be composed of separate nodules, and it could be recognized growing by the apposition of new nodules. The oldest portions were changed into apparently caseous detritus. Its new-formed blood-vessels entered also through Fontana's space and ran horizontally in a medial direction. Intra-ocular tension was reduced to minus three. It seemed beyond doubt that I had a granuloma to deal with. Since leprous granuloma does not occur in this country, and as specific granuloma could be excluded, and non-traumatic simple granuloma could not be considered, the diagnosis was reduced to tubercular granuloma.

In December, 1893, the right eyeball spontaneously perforated and a considerable amount of pus escaped, followed by a collapse of the globe. A careful examination of the pus for tubercle bacilli gave negative results. The opening rapidly closed, but intra-ocular tension remained reduced to minus three for several days, when it gradually rose. The bag-like elevation protruded again, fluctuated, and reburst in a similar place during the same month. No tubercle bacilli could be found in the discharged pus. In January, 1894, the opening closed, and intra-ocular tension increased. The eyeball became more consistent, and seemed to be filling gradually from behind by a more solid substance. Finally, during the second half of the same month intra-ocular tension became supranormal, the eyeball rapidly grew larger, so that the lids could not cover it, and it protruded for two centimetres from the palpebral fissure. At the same time rapidly increasing ocular pain developed. From the repeated unsuccessful though careful examinations for tubercle bacilli and the unexpected symptom-complex which appeared, I began to doubt the correctness of the first clinical diagnosis, and to think of the possibility of the presence of a glioma. This possibility changed into a daily growing conviction, until I enucleated this eye early in the following month.

Anatomical examination showed an ovoid eyeball with a longitudinal axis of forty-three millimetres and the longest transverse axis of thirty millimetres. The sclerotic was thin. The interior of the eyeball presented one continuous cavity, in which not a trace of iris, chorioid, retina, or lens could be found, but which was densely packed with fresh, well-stained glioma cells. In the outer equatorial region the sclerotic was ruptured, and the gliomatous mass had passed through this opening and

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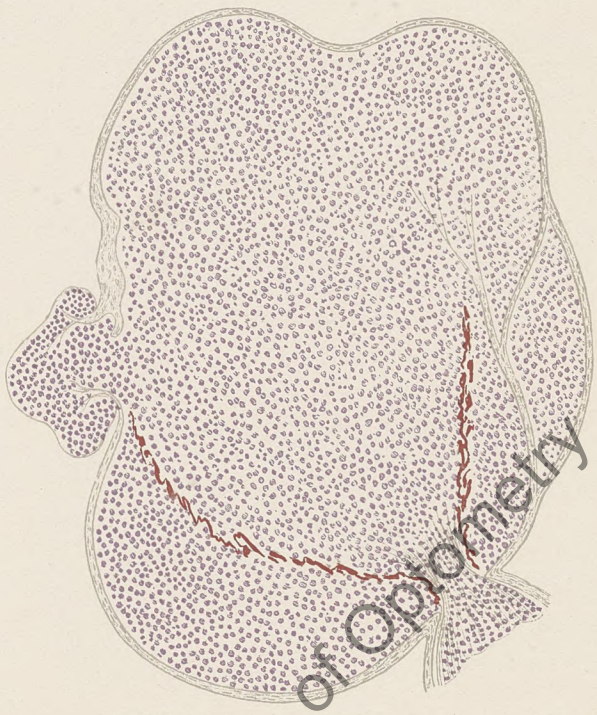
PLATE XXVI.



Section of eyeball showing crypto-glioma.

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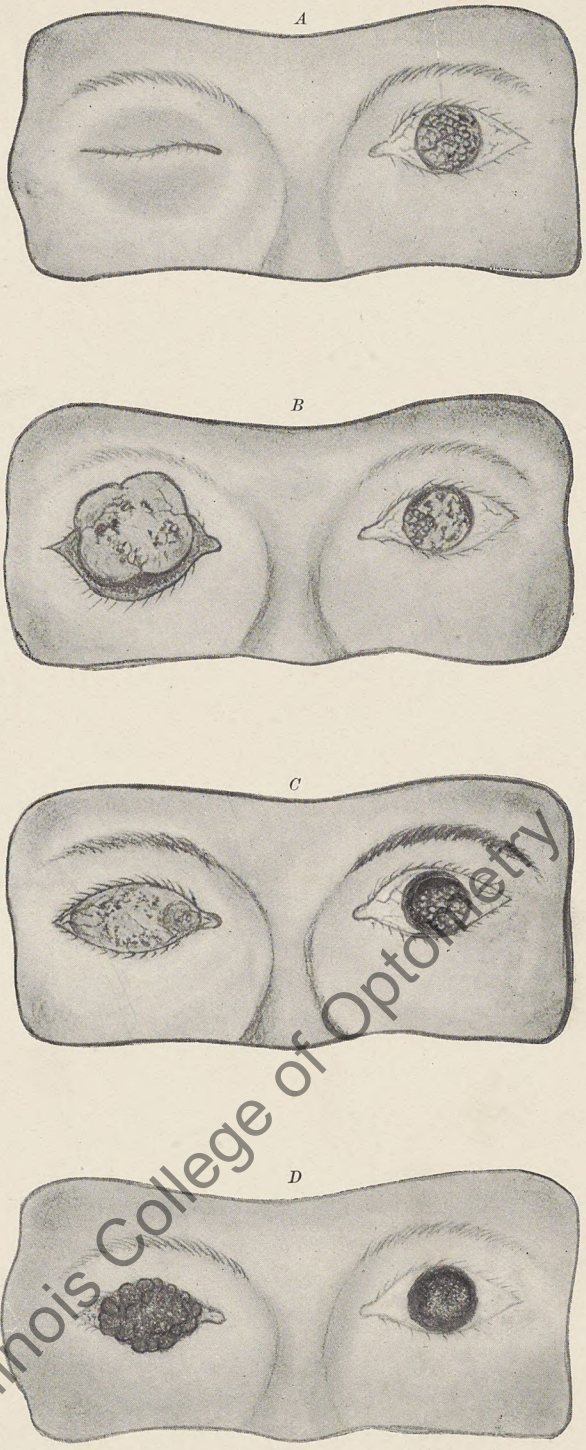
PLATE XXVII.



External appearance of glioma on the two sides.

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PLATE XXVIII.

Appearance of head of patient with glioma.

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formed a sharply defined epibulbar tumor of mushroom shape, eight millimetres broad and five millimetres high. On the medial side the sclerotic was split and the glioma cells were enclosed between its fan-like diverging fibres. Traces of the pigment epithelium formed a funnel which ran midway through the gliomatous material from the optic nerve to the ciliary body. The optic nerve was greatly thickened and was densely infiltrated with fresh glioma cells. The anatomical diagnosis, unlike the clinical one, was retinal glioma. (See Plate XXVI.)

The granuloma in the left eye grew slowly but steadily until it filled the whole anterior chamber. The regressive metamorphosis on the lateral side progressed until the tumor succumbed to it and the chamber became filled with detritus. At this stage a new tumor grew from the medial side of the eyeball into the anterior chamber. This stage is depicted in Fig. A of Plate XXVII.

At the demand of the parents, I had to discharge the patient early in February. Before this time the enucleation wound of the right side had healed. No local relapse occurred. In the left eye a similar granuloma sprang from the inner side. At first it formed a tongue-shaped prominence (seen in Fig. B of Plate XXVII.) which projected from the iris-angle and was composed of similar nodules. It had the same variety of new-formed blood-vessels. Increased by the apposition of new nodules, it finally filled the whole anterior chamber, the detritus having disappeared. During this period slight hemorrhages repeatedly took place.

In May of 1894 the patient was brought back to me. In Fig. C of Plate XXVII. I have shown the ocular condition at the time of discharge, while in Fig. D of the same plate the conditions found at the return of the patient can be seen. There was a local relapse on the right side, filling the orbit, the mass being barely covered by the lids. The left eyeball was somewhat enlarged, and intra-ocular tension, which during March had been minus three, had become more than normal, and the eye was painful to the touch. The anterior chamber was filled with reddish-brown and black masses, which were apparently composed of blood and hæmatogenous pigment. Later the anterior portions of the globe became increasingly ectatic, until an induced keratitis changed the cornea into a purulent mass, which the tumor pierced, forming an exophthalmic fungus.

Considering further operative interference useless, I abandoned the child to its fate, and tried by symptomatic treatment to ease its sufferings. Since there could no longer be a doubt as to the gliomatous nature of the affection in both eyes, not much that was interesting occurred between this time and that of the patient's death. The relapsing mass, as well as the exophthalmic tumor, grew steadily, causing recurrent hemorrhages. The patient became continuously weaker. The tumor finally reached the size of a man's fist and the primary growth that of a small apple, when, in November of the same year, death put an end to the patient's sufferings. Plate XXVIII. gives the appearance of the head of the patient at that time.

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I was unable to get either the tumors in their entirety or the head, barely succeeding in securing small particles of both tumors. Microscopic examination showed that the little pieces consisted of pure gliomata.

Von Graefe considers crypto-glioma due solely to inflammatory processes in the uveal tract as plastic irido-cyclitis, or subacute or chronic panophthalmitis induced by the glioma. Virchow believes that the temporary shrinkage of the eye in some forms of glioma is produced by a high-grade regressive metamorphosis and the absorption of the fluid elements. Personally, I think that both of these factors are active in most cases of crypto-glioma,—namely, induced inflammations of the uveal tract and tendency of the tumor to undergo regressive metamorphosis.

The crypto-gliomata run their course under three clinical pictures,—namely, plastic irido-cyclitis with pupillary occlusion, chronic or subacute panophthalmitis, and tuberculosis of the ciliary body and the chorioid.

I cannot agree with the statement of von Graefe that it is easier to diagnose a glioma in a phthisical stump than it is during its initial stages by means of the ophthalmoscope. Neither can I accept the points given by him in order to recognize glioma in a shrunken eyeball. He gives as two reasons the shape and the painfulness of the stump. He says that such stumps are always flattened antero-posteriorly, or that there is at least anterior bulbar phthisis. Yet such flattened stumps and anterior phthisis are observed with especial frequency with chorioiditis or retinitis or chronic purulent chorio-retinitis after cerebro-spinal meningitis, acute exanthemata, and penetrating foreign bodies. Moreover, usually in such cases the symptom-complex of the amaurotic cat's eye can be found, and a glioma can be easily falsely diagnosed where there is none. The painfulness, too, is not marked in glioma, since when there are lime deposits in such a stump, or bone plates in the chorioid, there may be more frequent and more intense pains than with glioma, while a stump containing a glioma may be painless. From all this it is plain that there are certain rare cases of glioma in which it is an impossibility to make a correct diagnosis, which are in truth, and probably will forever remain, crypto-gliomata.

The term pseudo-gliomata is or should be applied to cases in which the diagnosis of glioma has been made by a competent physician, while the later clinical course of the affection or the anatomical examination of the enucleated eyeball shows a different disease. Therefore a pseudo-glioma is equivalent to a mistake in diagnosis, and it is clear that there may be affections of the eye which bear a great likeness to the clinical picture and course of a true glioma. Pseudo-gliomata, consequently, may be defined as a group of heterogeneous eye-affections which run their course under the typical clinical picture of glioma.

In former times, when the symptoms of the amaurotic cat's eye in Europe were considered to be almost identical with glioma, pseudo-gliomata were quite frequent. In 1817 the name of amaurotic cat's eye was introduced by Beer, but, according to Hirschberg, it was probably intended to

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designate diseases other than glioma. Three years later Scarpa employed it similarly. Jaeger was the first to identify it with glioma, and Bauer, in 1830, considered the golden-yellow reflex from the interior of the eye as pathognomonic for glioma of the retina. Sichel and most other observers agreed with this belief. From this idea, the opinion obtained that glioma could be cured, and it is not astonishing that in those days most cases of glioma were said to be cured,—conditions which in reality were pseudo-gliomatous in type. This opinion was so firmly rooted that some even believe in a spontaneous cure of glioma, and Virchow does not consider it impossible. Hirschberg has sifted these cases critically, and has proved that in all probability they were examples of pseudo-glioma.

In 1886 Da Gama Pinto described an interesting case of pseudo-glioma in a boy three and a half years old, in which glioma had been diagnosed. Anatomical examination, however, revealed a funnel-shaped detachment of the retina with hyperplasia and a cyclitic membrane. Noyes and Jones have both described pseudo-gliomata of traumatic origin. Pseudo-gliomata have been reported by Collins, Rumschewitsch, Ginsberg, Cramer and Schulze, and others. I have described four cases, two of which proved to be chronic purulent chorio-retinitis and the other two hyperplastic chorio-retinitis of traumatic origin.

Pseudo-encephaloid is not identical with pseudo-glioma. The name encephaloid, as applied to-day to the malignant tumor of infancy called glioma, was introduced by Laennec into French ophthalmology, and has been accepted by Sichel, Robin, and most of the French ophthalmologists. Sichel believed that encephaloid was identical with our glioma, and that it meant a malignant tumor of the retina. It would appear to be logical that the term pseudo-encephaloid should be identical with our pseudo-glioma. This, however, is not the case.

After Langenbeck had correctly observed that these tumors spring from the granular layer of the retina, and represent, so to speak, a hyperplasia of this tissue, Robin made a similar observation in a case of intra-ocular growth, and thought he had to deal with a form of neoplasm differing from encephaloid,—namely, an innocent hyperplasia of the retina. On this observation Sichel based his idea of a benign encephaloid which was distinct from a malignant one, and strove uselessly to arrive at a differential diagnosis between the two. As the two were one and the same kind of neoplasm, this was, of course, impossible. By these teachings of a pseudo-encephaloid great confusion was produced in the chapter of glioma, which continued to quite recent time.

Glioma has been observed wherever competent ophthalmologists have been located. In the different European countries it seems to occur with equal frequency. Its occurrence relative to eye-affections in general, according to statements by Arlt, von Graefe, and others, is one case in from two thousand to two thousand five hundred,—that is, about one-half of one per cent. Since 1862 I have seen considerably more than a hundred

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gliomata among about two hundred thousand patients, which gives nearly the same percentage. The age at which it is seen varies between fœtal life and the twelfth year. Most of the cases, however, occur between the first and the third year. Congenital gliomata are usually bilateral. Gliomata have been observed in children of the same family. The number of non-congenital binocular gliomata is about ten per cent. of all the cases. No predisposition or other etiological factors are known. Although often maintained, a connection between trauma and glioma has never been proved.

Glioma almost always leads to death. The prognosis of operations for glioma, too, is unfavorable, since permanent cures are extremely rare. In former years the result of operative procedures was bad, probably, however, because the disease was not recognized as early as at present and hence was operated on at a too advanced stage of development. Even Wardrop, whose statements are true to this day, declared the disease to be incurable, but nevertheless he advised operation at an early stage. Middlemore, too, who was certain that the neoplasm was confined to the retina, recommended its removal by a plan somewhat similar to evisceration, in order to obviate enucleation. Most of the authors of their time, like Syme, Dalrymple, Travers, Scarpa, Weller, Rosas, etc., opposed the operation, being of the opinion that the patient's condition was made worse by the operative interference. These views on operations were followed by the observations of Langenbeck, Sichel, and Robin, which produced a reversion to the other extreme, and the prognosis was considered in too favorable a light. Through the labors of Hirschberg and von Graefe, however, this idea of the benignity of glioma has been corrected and Wardrop's stand-point—namely, that the prognosis of the operation is very unfavorable unless it is performed at an early stage of the affection—accepted. The number of well-authenticated observations of permanent cures of glioma by operation is practically still a small one, so that Da Gama Pinto in 1886 could report but fifteen instances. Personally, I should term the prognosis for operation favorable in cases in which a diagnosis has been made before a third of the vitreous space is filled by the tumor, unless the optic nerve is already heteroplastically diseased. I consider it a very doubtful procedure in case the eyeball is filled by the glioma, and I deem it particularly unfavorable in cases in which the mass has escaped from the interior of the eyeball.

As soon as the diagnosis of glioma has been ascertained, there can be no other indication but to remove the neoplasm as early and as completely as possible, with the eyeball itself. Enucleation is sufficient in the early stages of glioma when the growth has not filled half of the vitreous space. Even then, however, in these cases it is advisable to remove as much of the optic nerve as possible, since it cannot be ascertained whether this portion is heteroplastically implicated or not. At once after the operation the optic nerve should be examined, and if any pathological processes be found, the piece remaining in the orbit should be removed. In order to obviate this after-operation, which is neither easy nor without danger, von Graefe

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advised to combine at once a neurectomy with the enucleation. This he did by pulling the eyeball forward with forceps after cutting the tendons and entering a neurotome along the external orbital wall and cutting the optic nerve close to the optic foramen.

When the eyeball is filled with gliomatous tissue and it may be suspected that dissemination into Tenon's space has taken place, the extirpation of the eyeball, together with all the adjoining orbital tissue, is indicated. When there is a fungous exophthalmos, or when the gliomatous tissue has entered the orbit, exenteration of the orbit must be performed. In cases which have progressed further, removal of the eyelids must be added to the radical procedure. Eventually it may be necessary to remove some parts of the contiguous bones, followed by active cauterization. When the neoplasm has entered the neighboring cavities, especially the cranial, and produces cerebral or cachectic symptoms, operation is contra-indicated. In inoperable cases which must be left to their fate, symptomatic treatment may be employed to ease the patient's suffering. Antiseptics and narcotics are here of value.

Greeff has examined fresh gliomata by means of the impregnation methods taught by Golgi and Ramón y Cajal for the cerebro-spinal nervous system. By the latter method he succeeded in demonstrating the presence of differently shaped cells in retinal glioma. He found numerous oval and star-shaped cells with free offshoots which were undoubtedly true neuroglia cells, and which were the original cells of the tumor. The studies, furthermore, proved the presence of fibrous or apparently granular intercellular substances, most of which resembled embryonic neuroglia cells. He also found ganglion cells of different sizes, which were, however, not as frequent as the neuroglia cells. Small cells with one offset, resembling the embryonic retinal cells, the neuroblasts of His, could also be recognized ; these he believed to be embryonic ganglion cells. From these investigations it would appear that gliomata practically consist of hyperplastic glia cells, the offshoots of which form a dense net-work of fibres, ganglion cells, and nerve-fibres ; for this he proposes the name of neuroglioma ganglionare. The original views of Klebs are thus confirmed by these observations.

Of the theory of the spreading of glioma from disseminated germs (after Cohnheim, and enlarged by Greeff) not the least objective proof has been brought forward. Yet this paper of Greeff's makes it certain that gliomata are not neoplasms of a sarcomatous nature, since the impregnation of the cells after Cajal's method could not take place. The stained cells in gliomata are therefore all of a gliomatous or a nervous character.

XXXI. ENTOZOA.

In 1855 a case of *subretinal echinococcus* was reported by Gescheidt. As I know from his other publications that this author was a reliable observer of objective conditions, and as this case was quite a clear one and

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was exhaustively reported, I feel sure that it cannot be ignored, as has so far been generally done, for the mere reason that in recent times no similar case has been observed. I am convinced of the correctness of the description, and will report it here, without adding any critical remarks. "The eye was that of a pupil in a school for the blind. He was twenty-four years of age. During life a slight prominence of the organ had been observed, tension was increased, the pupil was irregular, there was partial cataract, and a yellow reflex came from the fundus. The patient died from some internal affection." As to the anatomical conditions of the eyeball, he reports as follows. "The space between the chorioidea and the detached retina was filled by a white bladder that was recognized as an echinococcus. This started from the lower surface of the retina, was bent around it, and filled the space between the parasite and the retina in such a manner that its two bag-like ends met above. Its outer skin was white, slightly translucent, and moderately firm. When it was opened, a little serous fluid escaped, followed by the appearance of a second bluish-white skin that was enclosed within the former. When this, too, was opened, some serous fluid which contained a large number of small, round, oval, or olive-shaped worm-like bodies that rested upon the inner surface of the thin skin was evacuated. Some of these placed under the microscope showed undoubted small round suction openings. Hooklets could not be found."

Cysticercus Cellulosæ.—The first cases of cysticercus in the depths of the human eye were recorded after the discovery of the ophthalmoscope by Coccius in 1853 and by von Graefe in 1857. At the stage of invasion, the subretinal cysticercus forms a small loose detachment of the retina, on account of which, since the disturbance of the detached part of the retina is not important, a bluish oval bladder from two to five millimetres in diameter, on which, under favorable circumstances, and especially at its margins, peculiar interference colors can be observed, may be noticed. Occasionally the head segment can be differentiated as an intensely white spot; and, favored by fortune, one can observe its movements in connection with peristaltic motion of the bladder, or, when exceptionally fortunate, independent forward extension of the head segment may be recognized, allowing a correct diagnosis to be made. Sometimes a subretinal cysticercus can be seen moving between the retina and the chorioid, and later, becoming larger, it may be observed breaking its way through the retina into the vitreous humor. The original position of the cysticercus beneath the retina and the route which it has gradually made often remain visible for long periods of time.

After recent migration of the cysticercus the disturbance of vision must be very slight, especially if the entozoon has assumed an eccentric position. On the other hand, in case of a central position of the cysticercus in the region of the macula, severe disturbance of vision will soon cause the patient to seek aid. Frequently patients assert that they see a spherical shadow. A patient of Hirschberg's described quite correctly the entoptic image of

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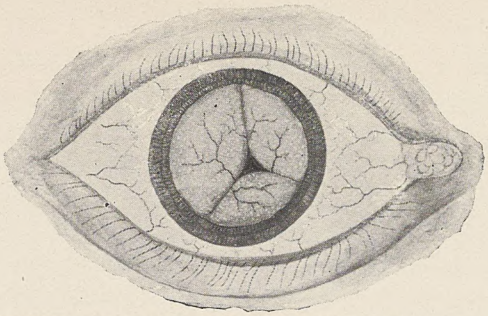
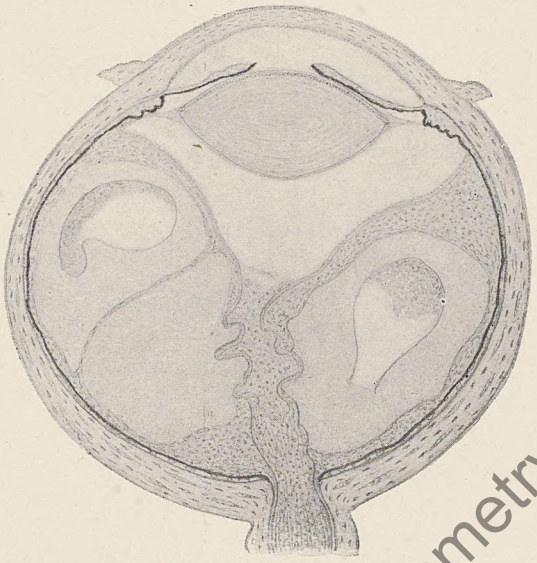
PLATE XXIX.



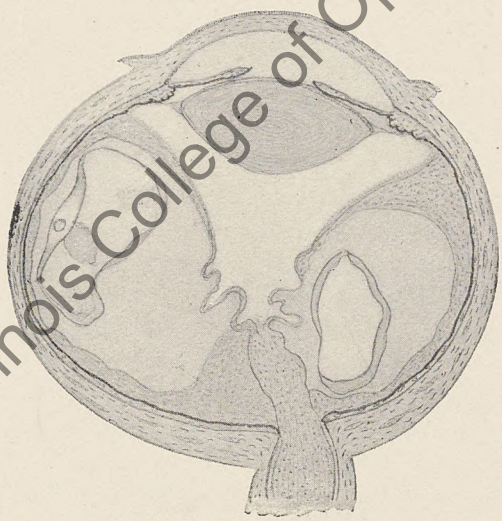
Appearance of subretinal cysticercus under focal illumination.

PLATE XXX.



Section of subretinal cysticercus.

PLATE XXXI.



Section of subretinal cysticercus.

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the cysticercus, first with its head projected and then with its head withdrawn. These defects in the field of vision increase in the course of time, till, after the lapse of months, the faculty of seeing is limited to quantitative light-sensations, and finally is lost. In these cases the eyeball itself is often found to be shrunken or atrophied. In some types the entozoon remains beneath the retina, in others it is situated before the retina, and rarely it will be found between the retinal elements. A dense cloudiness of the vitreous humor, especially of a membranous nature, together with shrinkage and thickening of this humor, has been found in nearly every instance. In most of the cases the retina has become detached, the detachment being usually total. As a rule, the retina is degenerated, often of a cystic type. Sometimes degenerative changes in the chorioid have been shown, though rarely bone-formations take place. The ciliary body often appears detached from the sclera, the lens frequently becomes cataractous, the cornea is clouded, while the sclera, though rarely, becomes thickened and infiltrated with pus.

In May of 1892 I saw a unique case in a five-year-old boy. The patient, considering his age, was well developed and healthy. The right eye was normal. The tension of the left eye seemed somewhat increased. The pupil was not as wide as that of the other eye, and the iris reacted sluggishly. A whitish-yellow reflex could be perceived. After the pupil was dilated, and under focal illumination, three separate portions of the retina were visible, filling the greater part of the vitreous space like three elliptical bags. The largest of these bags lay outward and downward and stretched from the ora serrata to the posterior pole of the eyeball. The second, a medium-sized one, was situated inward and downward. The third was situated upward and a little less inward. The walls of these bags appeared yellowish white and were semi-transparent. They were sprinkled with small, point-like spots. The normal retinal vessels extended over the surfaces of the bags. Even rapid movements did not change the size, shape, and relative position of the bags, yet a slight trembling, such as might appear on a bladder not quite filled with fluid, could be seen on them. With strong convex lenses and intense illumination sufficient light could be thrown into them to enable me to perceive a large membranous body of elliptic shape with a silk-like lustre in each. I also saw granular, dim spots on these bodies. The patient felt no pain. Vision was almost abolished. I made the diagnosis of triple subretinal cysticercus. Plate XXIX. shows the appearance of the eye under focal illumination. Temporary symptoms of ciliary irritation appeared, the anterior chamber grew more shallow, and the iris became discolored and lost motility. The retinal bags were less transparent, so that the formations lying behind the retina grew steadily less visible. Tension increased, the media grew dim, and considerable pain appeared, so that in July of the same year I enucleated the organ.

Upon section, a subretinal, somewhat shrunken cyst appeared, which

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from the cuticular appearance of its walls was recognized as that of a parasite. On further section, two other cysts became apparent. In one of these secondary sections, which was purposely made thicker, a round opening, with a receptacle inward in which the retracted scolex was located, was found in the covering of the larger cyst. Thus the diagnosis was proved correct beyond a doubt, anatomically as well as zoologically. I did not succeed in demonstrating the other two scolices in an equally fortunate section, since the cysts were extremely friable. Later, however, I obtained some of their fragments from the fluids under which I had done the cutting. Plates XXX. and XXXI. show two of the best sections taken from different depths.

My own observations show that in Bohemia cysticercus is very rare. While serving as assistant at the von Hasner clinic, which had an abundance of material (since to this place came all the serious eye-affections from the whole of Bohemia), I did not see a single well-authenticated case of intra-ocular cysticercus from 1862 to 1865. It was not till 1870 that von Hasner observed his first case of intra-ocular cysticercus in Prague. During the time Professor Sattler had charge of von Hasner's clinic, two cases of intra-ocular cysticercus were observed, one in the vitreous space and the other in a subretinal situation. While Professor Schnabel was in charge not a case was seen, and at present, under Professor Czermak, not one has been found. From these statistics only three certain cases have occurred in this enormous and long-known clinic. Among the one hundred and thirty thousand patients whom I have so far seen in the different official positions that I have held, I have found only two undoubted cases.

The prognosis of intra-ocular cysticercus is bad. Not interfered with, the eye is always lost. The oft-cited case of Teale with a favorable end is, according to Hirschberg, based on a diagnostic error, probably being one of congenital malformation of the vitreous humor.

Medication as at first applied by von Graefe, such as instillations of potassium and santonin preparations in order to kill the entozoon, have proved unsuccessful. This has been the case also with the injection of other agents into the interior of the eyeball and the application of the electric current. His advice to kill the entozoon with a cataract-needle was not followed even by himself. De Wecker, however, tried to accomplish it with a von Graefe knife, under guidance of the ophthalmoscope, though without success. Hahn and perhaps Desmarres also attempted the same. In 1856 von Graefe performed the first cysticercus operation, having preceded it by an iridectomy made for better localization of the parasite. He opened the sclerotic with a broad, flat reclination-needle and enlarged the opening with a scissors. After some difficulty, he extracted the entozoon with a pair of capsular forceps. On account of the non-successes and dangers of the operation, von Graefe in 1858 attempted to make the extraction through the cornea. At first he did this in three acts,—namely, iridectomy, extraction of the lens, and extraction of the entozoon. Later he

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accomplished the three procedures in one sitting. In 1866 he adopted an equatorial incision, and in 1868 stated that this should be done in emergencies only, and that for ordinary cases the corneal extraction in one sitting was preferable. On the whole, the final results of von Graefe's operations were unfavorable.

Arlt was the first, in 1879, to introduce the meridional incision by which subretinal cysticercus might be removed. Alfred Graefe, in Halle, enlarged upon Arlt's method and brought it to its present state of perfection. He modified the incision by turning the sharp edge of a knife towards the sclerotic. As a result he soon published twelve cases with favorable issue. In 1882 he constructed a special ophthalmoscope for localization, and advised the plan of tattooing the meridian selected before operating. In 1878 Becker, Leber, and Cohn operated according to this method, each having one case with good result. Cohn has advised to determine the position of the entozoon perimetrically before the operation. In 1885 Alfred Graefe reported sixty-seven per cent. of successes. In 1890 Vossius employed the narrow knife for subretinal cysticercus and the lance knife for cysticercus in the vitreous body.

From this it can be deduced that, especially since perfected by Alfred Graefe, the operation for intra-ocular cysticercus has, when early performed, a favorable prognosis. However, when the parasite has once produced material destruction and more or less severe inflammation has arisen, the operation for removal of cysticercus is no better than enucleation.

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DISEASES OF THE OPTIC NERVE.

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PART I.

CIRCULATORY AND FUNCTIONAL DISTURBANCES AFFECTING THE PAPILLA OF THE OPTIC NERVE.

THE supply and distribution of blood in the papilla of the optic nerve are mainly dependent on the vascular system of the retina; therefore this subject will be found more fully treated in considering the circulation and the metabolism of that structure. It may add to the completeness, however, to give in this place a brief recapitulation of the more important points.

Pulsation of the main venous branches of the papilla of the optic nerve occurs so often as a physiological manifestation that little diagnostic value can be attached to it unless the presence of the venous pulse is accompanied by a history of its previous absence. Normally, or after the inhalation of nitrite of amyl, venous pulsation will be found to correspond to the heart's diastole. Abnormally, this pulsation is not only more noticeable,—as in aortic insufficiency without compensatory hypertrophy,—but also varies as to the time of its appearance, beginning, for instance, during the heart's systole in tricuspid insufficiency.

Arterial pulsation, which is normally absent, is of much greater diagnostic importance. This condition is most commonly met with in aortal insufficiency, although the rhythmic reddening of the whole area of the optic disk, known as the "pulse" of the papilla, can seldom be detected. Arterial pulsation is much more rarely seen in cases in which the blood-pressure is greatly reduced, as in extreme anæmia. It is also met with, although very infrequently, in aneurism of the aorta. At times, when the aneurism is near the origin of the left carotid, more marked pulsation of

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the arteries upon the left papilla may be discovered. In addition, the arterial pulse has been noticed in Basedow's disease. It is in increase of intra-ocular pressure (glaucoma), however, that pulsation of the arteries attains special significance. This disease produces a bounding pulse, similar to the effect caused by pressing the finger on the wall of the globe, the blood being driven into the arteries by the systolic impulse, and being forced out again almost immediately by the intra-ocular pressure. Attention is likewise directed to the rhythmic filling and emptying of the venous channels, even when the tension is normal, in cases where the central artery of the retina is compressed,—either directly behind the retina or even at a considerable distance behind the globe.

The most common disturbances affecting the circulation of the optic nerve are:

1. Anæmia of the papilla.
2. Hyperæmia of the papilla.
3. Hemorrhage in the papilla.

ANÆMIA OF THE PAPILLA.

As a local manifestation of general disease, anæmic pallor of the optic disk is seen in chlorosis. In the various forms of cachectic anæmia, in addition to pallor, it is comparatively common to find the signs of grayish discoloration of the papilla, together with slight blurring of the borders; in exceptional cases, the beginning of swelling and œdema of the disk may be noticed. In pernicious anæmia and leukæmia,—a waxy, yellow pallor being characteristic of the latter disease,—the vessel-walls are somewhat contracted, while the vessels themselves look strikingly pale in color, this being due to the excess of white blood-corpuscles and the scarcity of hæmoglobin.

In all the diseases mentioned, general hemorrhages—as a rule, with a whitish centre—are repeated and profuse, and similar extravasations of blood are occasionally discovered upon the papilla and along its margin. Pathological examination has even established the presence of thrombi in the central vein of the retina.

Anæmia of the papilla has also been reported as occurring in syncope. The writer's experience does not warrant him in confirming the appearance of a perceptible pallor in ordinary attacks of fainting, such as accompany, for instance, the removal of a foreign body from the cornea. At most, the arteries may undergo slight contraction; the veins, on the contrary, seem somewhat dilated.

Pallor of the optic disk is of greater prognostic significance after excessive hemorrhage or when it is associated with quinine intoxication, as in the so-called ischæmia of the retina. (See Atrophy of the Optic Nerve.) After ligation of the common carotid, immediate emptying of the retinal artery of the same side results.

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HYPERÆMIA OF THE PAPILLA AND THE SO-CALLED "NERVOUS HYPERÆMIA."

Hyperæmia of the optic disk may show itself:

A. *In General Diseases.*—In grave congenital cardiac lesions, more rarely in acquired heart-disease, all the blood-channels of the papilla, but particularly the veins, may undergo perceptible dilatation, with the result that the optic disk, and also the vessels themselves, present an almost bluish-red color. Knapp, in 1870, described an intricate ramification and enormous distention of the retinal vessels (Medusa head). A moderate degree of congestion may show itself in secondary stages of syphilis, while a high degree of hyperæmia is commonly found in advanced cases of diabetes, even though no definite lesion of the optic nerve or retina can be detected.

B. *In Local Ocular Lesions.*—Exceptional dilatation of the veins, with numerous hemorrhages scattered throughout the retina and even surrounding the papilla, can often be detected in thrombosis of the central vein of the retina. Hyperæmia, in addition, shows itself upon the papilla during irritation and inflammation of the cornea and in the various inflammatory conditions of the ocular tunics,—cornea, sclera, ciliary body, and chorioid. If the vitreous humor is turbid, the papilla, for evident reasons, shines through like a dusky red disk. Dilatation of the veins and disappearance of venous pulsation, if the latter formerly existed, are also noticed after compression of the jugular veins, as well as during forcible expiration, and after elevation of the upper extremities.

C. *In Nervous Disorders.*—Distention of the veins and arteries and smaller vessels of the papilla, with resulting hyperæmia, may be observed after the fatigue of tedious railway journeys, after enforced wakefulness, after prolonged use of the eyes, after excessive alcoholic indulgence, and after the inhalation of nitrite of amyl. Congestion of the disk is seen with especial frequency during the stage of delirium in psychosis. The writer cannot, however, confirm the statement that a hyperæmic papilla is particularly common as an attendant symptom in insanity. Nor can he agree with the statement, repeatedly made, that serious middle-ear disturbances are often accompanied by hyperæmia of the optic nerve. On the contrary, the papilla is so rarely congested, even in cases of lethal meningitis and cerebral abscess of otic origin, that no conclusive inference concerning the gravity of the aural lesion can be drawn from the appearance of the disk.

D. *In Functional Disturbances.*—It is in functional neuroses, hysteria, neurasthenia, and nervous asthenopia, as it has been called, that hyperæmia of the papilla deserves particular consideration. These cases are very frequently met with in practice, and the patient's statements are so misleading that inexperience may lead to a wrong diagnosis or to a hasty expression of opinion, with the result that the existing manifestations are merely aggravated. This disturbance usually appears during youth,—chlorotic,

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pale-faced girls at puberty being especially affected,—although it may occur in later life, as in women at the menopause. Pressure in and about the eyes, pain in and behind the globe, and frontal and temporal headaches, constitute the usual symptoms, together with complaints in almost every case regarding "muscæ volitantes." These objects are described in detail, and their varying shapes and gyratory excursions studied and pursued with such intentness that a feeling of dizziness is sometimes produced. At other times a lancinating pain shooting through the eyes is designated as "terrible;" then, again, the patient complains that he finds it impossible to look at people in general or at any person in particular, or to fix any object, without experiencing suffering. Less frequently it happens that the patient sees in the dark, or with eyes closed, semicircular or circular particles of light (phosphenes); or in very rare cases he states that objects seem covered with colored designs. The most common complaint, however, is the fear of being overtaken by one or all of the serious diseases of the eye that the patient may have seen or heard of.

Although in the majority of cases hysterical paroxysms—paresis and hemianæsthesia, etc.—are absent, the possibility of their appearance cannot be excluded. Some patients complain of fluctuation and a feeling of fluidity in the brain, with pain and a crackling sensation in the nape of the neck and in the occipital region. During the menopause, wave-like gushes of heat make the sufferer feel as if hot water were being poured over the entire body. These and other psychical phenomena may often be traced to a remote origin, such as some serious injury or accident, blighted hopes, or sexual unrest and excesses.

In the nervous asthenopia prevalent among school-children, the child generally complains that the print looks blurred and that the letters are intermingled and the spacing is irregular. This is soon followed by luminous rings, and little by little the patient approximates the book to his eyes in order to obtain (as in accommodative asthenopia) the largest possible retinal image. Undoubtedly, in some of these children, the constant thinking of weak eyes or hearing of diseases of the eye, together with noticing that other persons wear glasses with seeming benefit, may be responsible for much of the disturbance by producing auto-suggestion and imitation. In most instances, however, children of this class are burdened with feeble constitutions and neuropathic legacies. They are generally quiet in disposition, but not dull mentally. Often, too, other neurotic evidences manifest themselves; among these are clonic contractions affecting the muscles of the face or the extremities, pseudopia, and visions (hallucinations). For example, the child may see heads and fantastic shapes in the window, but the fact that the patient admits that the appearances in question are illusory and unreal makes the differentiation from true hallucinations easy.

In many of these neurotic and hysterical subjects, vibratory contraction of the upper lid as soon as the patient is asked to shut the lids gently is a characteristic symptom, and often a striking oscillation of the globe follows

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every attempt on the part of the physician to inspect such eyes. A less certain symptom is the anæsthesia of the ocular conjunctiva and cornea so often referred to. A similar lack of reaction to excitation may be observed in indolent but otherwise healthy individuals.

Another neurotic expression, which Krafft-Ebing describes in masturbators, is sometimes seen in boys and young men, and consists of a peculiar lustre and reflex of the cornea. This is due to the greater exposure of the globe on account of the marked retraction of the upper lid. It is not at all unlikely that this retraction is dependent on increased innervation of the sympathetic fibres that control the muscle of Müller, in this way intensifying the action of the levator muscle of the upper lid. It is probable that the pupil appears wider and broader for the same reason. It has been noticed, in addition, that cocaine produces more rapid and greater pupillary dilatation in these cases than in healthy subjects.

It must be remembered in all these cases that a disease trifling in itself, or an anomaly of refraction that is too slight to account for the degree of disturbance complained of, may, under certain environments and by reason of incessant introspection, develop into a serious lesion.

Hyperæmia of the conjunctiva, or "conjunctivitis sicca," and congenital punctate opacities of the posterior lens-capsule, may accompany the nervous disturbances mentioned. In some instances a moderate degree of myopia or astigmatism exists.

As regards ophthalmoscopic appearances, the optic disk in these neurotic patients discloses a typical picture. The papilla is scarlet red in color,—glistening and fiery,—presenting in the inverted image a color-effect that has been compared to that produced by the rising moon. In contrast to this, the physiological excavation, if it exists, stands out in striking whiteness. By the direct method, we can verify the anatomical finding that the nerve-fibres are more abundant on the nasal side of the papilla than elsewhere, as is proved by the fact that this portion of the disk is redder, sometimes discolored, and may even show blurred edges. The well-known striation and mottled appearance of the nerve-fibres in their passage over into the retina will be found accentuated, and this condition is even more pronounced at the boggy and hyperæmic margin of the papilla and on the adjacent retinal surface. If only one eye is affected, this deep redness of the optic disk is more marked than in the other eye.

The patient usually states that his vision is impaired both for distance and close at hand. This assertion cannot always be verified; in those instances where vision is said to be reduced to one-third, one-half, or even less, a condition is reached which borders on hysterical amblyopia and amaurosis. The consideration of such cases will be found in a special chapter. In a favorable case, however, where a patient declares that his vision is defective but where all indications point to emmetropia or moderate hypermetropia, it is a good plan to place before his eyes weak convex and concave lenses of the same strength. Not knowing that he is looking through a plain

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glass, and concluding that lenses ought to improve his sight, the patient almost invariably allows himself to reach the normal standard of vision. This method is particularly successful in cases of nervous asthenopia. These neurotic patients often wear weak convex or concave glasses, procured of their own accord or as the result of a faulty diagnosis on the part of a physician.

A trustworthy aid in diagnosis is the perimeter. It is by no means always necessary to test every meridian in these cases: the horizontal is generally sufficient. In conducting the test, the first requisite is to comprehend fully the principles of perimetric methods. Foremost among these is that the examiner concentrates all his attention upon the cornea of the patient, so that he is made aware at once of any sudden and often unconscious rotation of the globe and any other movements of the eye which would necessarily prevent accurate results.

As is well known, the eye in its normal state may be said to recognize colors in squares (5 mm.) at a peripheric distance which for practical purposes may be approximately given as follows:

White,	temporal side, (almost)	90°—80°;	nasal side,	60°—50°.
Blue,	"	60°—50°;	"	40°—30°.
Red,	"	50°—40°;	"	40°—30°.
Green,	"	35°—40°;	"	30°—45°.

In the neuroses under consideration, a variation in the above limitations generally assumes one of three expressions: first, a general contraction for all colors up to ten towards the point of fixation; second, the contraction affects only the limits for blue, green, or red; or, third, the colors change their usual relationship, so that from the periphery red (or more rarely green) is recognized in advance of blue (or even white). The so-called phase of exhaustion mentioned by Förster can be determined with comparative readiness. The longer the perimetric examination is continued, the more contracted the limits of the field become. In many cases it is evident that, in order to be convinced that the inaccuracies found are not due to inattention on the part of the patient, it is necessary to make carefully regulated and repeated tests.

In the differential diagnosis, contraction of the field of vision might suggest retinitis pigmentosa, but only that form in which the equatorial region and the periphery are not much affected: if the characteristic pigmentation is present, a mistake is hardly possible. Besides this, in pigment-degeneration of the retina, central acuity of vision is seldom impaired, except in advanced cases or where the later degenerative process, consisting of a stellate opacity of the posterior cortical layers of the lens, has appeared. The history of heredity (present in most cases), the disordered light-sense, night-blindness, etc., also aid in the diagnosis.

The distinct discoloration of the disk (at times involving the margins of the papilla) which is met with in both intra-ocular and retrobulbar

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neuritis will serve to differentiate the ophthalmoscopic appearances found in these diseases from the picture presented by simple hyperæmia of the papilla of functional origin. More than this, in incipient intra-ocular neuritis there are present intense headaches, not relieved by occupying the patient's attention with other matters; also vomiting, vertigo, etc. These symptoms may precede for months any disturbance in visual acuity, if vision is affected at all,—since it often happens in cerebral tumors that sight is for a long time in no way impaired. In retrobulbar neuritis the presence of a central scotoma can likewise be established; in many cases of brain-tumor, hemianopic disturbances of the visual field appear; both of these conditions are absent in nervous hyperæmia of the papilla.

Furthermore, the fact that in exceptional instances a shining, striking redness of the papilla, dependent on the large number of minute blood-vessels, may constitute a congenital anomaly, especially when the fundus oculi is dark in color, must not be lost sight of.

The prognosis in nervous hyperæmia of the papilla and at the same time for the accompanying disturbances is, almost without exception, favorable. With suggestive treatment, the suffering ceases at once or after a few days. Kopiopia hysterica may occasionally prove an exception to this rule and may persist for months. Recovery is materially hastened by avoiding any reference to the disease which affects these neurotic cases. The use of expressions such as "congestion of the optic nerve," "hemorrhage," etc., is certain to make the patient unnecessarily fearful and apprehensive.

Mild collyria may be prescribed for the relief of the conjunctivitis. Compresses dipped in Burow's solution and bathing the eyes with a two per cent. solution of boracic acid ordinarily suffice. In some cases it may be advisable to prescribe lenses (0.50 to 1 D.), with the injunction that they be worn for a short time only. The patient will soon abandon the use of the glasses of his own accord, since they can afford only temporary relief by suggestion.

HEMORRHAGE IN THE PAPILLA.

The seat of hemorrhage in and around the optic papilla is between the nerve-fibres. Since these take a radiating course, we can understand why the outline of the hemorrhages is so commonly radiating and flame-like. Such extravasations of blood accompany most of the hemorrhagic lesions of the retina, and are fully described in the part devoted to this subject.

Here, however, only those almost isolated hemorrhages that occur in the papilla itself are considered. These extravasations are usually the results of grave cardiac lesions; they may be met with at any age, but are more common during middle life, and occur more frequently even during advanced life, as an expression of arterio-sclerosis. They are generally unilateral. As a necessary sequence of the cardiac lesion, the ophthalmoscope discloses disordered circulation in both eyes, and in arterio-sclerosis peculiarly twisted, pale arteries are noticed, presenting a broad central band

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which appears puckered and torn in places, with here and there a shining reflection. The rupture itself may appear in segments or as a quadrant, or it may even occupy one-half of the papilla as a jet-black hemorrhage not sharply defined, and ordinarily projecting over the edge of the disk. The hemorrhage occupies a different level in the fibre-layer, which is quite dense in this situation, or even behind it. The patient states that there is a veil or a mist before his eyes upon awakening, the cause of which must have come on during the night; or he gives a history of having discovered the imperfection of sight by accident, some discomfort in binocular vision leading to testing the acuity of each eye separately. Vision is most interfered with if the temporal side of the disk—*i.e.*, where the fibres pass on to the macula—is attacked.

The prognosis is ordinarily quite favorable. Hemorrhage dependent on arterio-sclerotic changes is generally followed in the course of a few years by similar apoplectic foci in the brain. A description of the pathological changes affecting the walls of the blood-vessels in this disease will be found under "Diseases of the Retina."

As regards treatment, the avoidance of all unnecessary use of the eyes is demanded. This caution also applies to bright illumination. Rest in the recumbent position is desirable, but not imperative. Everything that is likely to increase the general blood-pressure or that tends to produce congestion or circulatory stasis must be prohibited (stooping, coughing, sneezing, constipation, the wearing of tight cravats, the use of alcoholic stimulants, etc.). In cardiac lesions without compensatory hypertrophy, digitalis, followed by a symptomatic, dietetic course of medication, is indicated. The use of moderate doses of potassium iodide, administered for three weeks, followed by suspension of the use of the drug for one week, often meets the requirements of the case and the expectations of the physician. To hasten absorption of the hemorrhagic extravasations, moist heat, warm fomentations, etc., may be employed; inunctions of iodine ointments are also useful when applied to the brow and temporal region, precautions being taken, however, that no eczema is excited.

It is admitted that it is often possible to make the diagnosis of arterio-sclerosis by the appearance of the retinal vessels. In this connection the writer desires to call attention to an original observation. In those cases in which rupture is imminent—either in the vessels of the retina or at times also in the papilla—or has already occurred, compression of the walls of the globe with the finger no longer excites pulsation in the degenerated arteries. The pulsation in question, as is well known, can be produced readily and in a marked degree, even in cases of choked disk. It is therefore not merely an expression of arterio-sclerotic contraction, but must rather be looked upon as an induration or rigidity of the walls of the arteries, which even increased tension produced by pressure with the finger during diastolic contraction cannot overcome. This observation applies in particular to arterio-sclerosis affecting the principal branches

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of the blood-vessels upon the papilla; it does not hold good for the arterio-sclerotic degeneration of smaller and isolated branches at remote points in the retina, even when rupture has taken place.

The writer has noticed the same phenomenon—inability to excite arterial pulsation when pressure is made against the globe in old persons—in several cases of thrombosis of the central artery of the retina. The main arterial channels upon the disk appeared like broad, straight, hyaline tubes of dirty-red or orange color; the vessels were accompanied by an uncommonly broad central band that presented a dull reflex. In their remoter ramifications over the retina, the arterial branches were filled with dark, imperfectly connected columns of blood that had passed the strangulated portion of the vessel. In addition, here and there in the neighborhood of the blood-vessel trunk the blood had exuded and extravasations in the form of stripes and spots could be detected. The venous channels, on the other hand, appeared slightly contracted, a condition which can be readily accounted for in view of the impediment offered to the entrance of arterial blood and the stasis in the arterial branches and the free outflow of blood from the veins. The papilla, whose margins were surrounded by a few hemorrhages, was of a deep cherry-red and looked as if it had been steeped in hæmoglobin. The vitreous humor showed diffuse haziness, and visual acuity amounted to bare perception of light. After three weeks, blindness resulted.

The writer has seen a case of embolism of the central artery of the right retina in a married woman who had repeated and numerous evidences of the formation of blood-clots. These masses could be felt in the superficial veins of both hands, on the dorsal side, and were slightly sensitive on pressure. The patient, being lightly clad and having no covering over her head, ran out into the garden on an autumn day and hurriedly began to rake together the fallen leaves. Almost immediately dark circles appeared before her sight, followed by total loss of vision in the affected eye. In this eye there were the ophthalmoscopic evidences of embolism of the central artery of the retina.

PART II.

INFLAMMATION OF THE OPTIC NERVE.

Inflammation of the optic nerve may affect,—1, the intra-ocular termination of the optic nerve or the papilla; 2, the retrobulbar portion, including that part of the nerve in the bony canal formed by the optic foramen; 3, the intra-cranial division.

Considered from the stand-points of pathology and morbid anatomy, this clinical classification seems somewhat arbitrary, since in the majority of cases of so-called intra-ocular neuritis both the retrobulbar and intra-cranial divisions of the nerves participate in the inflammatory changes.

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SUBDIVISION I.

INTRA-OCULAR NEURITIS.

GENERAL CONSIDERATION.

The general ophthalmoscopic picture in intra-ocular neuritis shows that the entire surface of the optic disk is discolored. The boundary lines are cloudy and indistinguishable, while the papilla itself assumes a turbid, dark-red tint. These alterations, together with the attendant hyperæmia and haziness, are least pronounced in the temporal quadrant and most striking and constant on the nasal side. The principal veins upon the papilla are somewhat distended; at the edge of the disk these vessels (either one or both) undergo a curvature which is manifestly more convex and abrupt than that usually noticed at this point. At the same time the blood-vessels in different parts of their course look hazy and blurred; this condition is not only met with near the excavation and internal to the bend mentioned, but even involves the venous channels external to this point,—that is, in the area bordering on the margin of the papilla.

If the direct method is resorted to, radiating striæ, arising most frequently from the upper and lower boundary of the disk and extending into the adjacent retinal space, can be detected. These markings indicate the transition of the nerve-fibres into the retina, and can often be detected in the locality referred to, even in the normal eye. The only difference is that the normal layer is more transparent throughout and its striation more delicate than in the pathological picture.

In addition, intra-ocular neuritis is often accompanied by special changes in the nasal portion of the papilla. The disproportionately large mass of nerve-bundles piled up here makes this a favorite focus for inflammation. The whole tract is not merely characterized by diffuse, dusky-red cloudiness, but often presents a mottled appearance with punctate and striate spots; more than this, the nasal side of the papilla may be found swollen, elevated, and projecting above the level of the surrounding retina. At times the smaller blood-vessels in the substance of the papilla are implicated and show striking engorgement and tortuosity.

The changes disclosed by the ophthalmoscope manifest themselves under two chief types:

1. Intra-ocular neuritis as a result of so-called retrobulbar neuritis, the inflammatory process being transmitted from the orbital division of the optic nerve to the papilla.

This is the more uncommon cause of optic neuritis, and its detailed consideration as well as the differential diagnosis from brain-tumors will be taken up in the chapter on Retrobulbar Neuritis.

2. Intra-ocular neuritis in the form of incipient choked disk.

This is the more common cause. The preliminary statement is here made that, in conformity with the opinion of most oculists (Uhthoff), swelling of

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the optic papilla is regarded and designated as "intra-ocular neuritis" as long as the distance from the apex of the swollen disk to the level of the retina does not reach three diopters. Only when this distance passes beyond three diopters is the process to be considered as so-called "choked disk." It does not seem expedient to modify this designation at present.

SPECIAL OPHTHALMOSCOPIC AND CLINICAL FEATURES.

Intra-ocular neuritis of the second division may be designated for the sake of brevity as "incipient choked disk."

The ophthalmoscopic appearances just described are found in brain-tumors, in tumors of the meninges and neoplasms of the cranial cavity in general, in cerebral cysticercus, and in inflammation of the meninges (tubercular and syphilitic meningitis). Less common causes are septic and epidemic meningitis, hydrocephalus and cerebral abscess, tumors of the orbit or of the orbital walls, neoplasms of the optic nerve, Bright's disease, superior polioencephalitis, hemorrhagic pachymeningitis, and occasionally chlorosis, emphysema, and cardiac lesions.

Tumors of the brain constitute the exciting cause in the largest percentage of cases.

The one symptom which is almost pathognomonic in nearly all the lesions mentioned is headache. Since this condition may often persist for weeks, and even for months, it is not merely expedient but necessary in all cases of continued and inveterate headache to make frequent and repeated examinations of the fundus of the eye. As soon as the changes referred to are discovered on the part of a previously normal optic papilla, the differential diagnosis between this and other varieties of cephalalgia is comparatively easy.

Headache is particularly common in the early stages of Bright's disease. The urinalysis, the demonstrable arterio-sclerosis, and the small circular hemorrhages seen in the retina ought to guard against errors in diagnosis even before the existence of albuminuric retinitis is definitely established.

Another form of cephalalgia is that arising in hysterical patients. This headache is apt to be exaggerated and described as "excruciating," although it can often be made to disappear by directing the patient's attention to other matters. Added to this, the state of the fundus, and the shining red optic disk previously mentioned, together with the characteristic field of vision and other evidences of hysteria, tend to suggest the proper diagnosis.

It is easy to differentiate the periodic attacks of sinus-diseases (in rhino-pharyngeal disturbances, bronchial affections, and influenza); in these cases, in addition to the history, local evidences of obstruction on percussion often make the diagnosis evident. It is also easy to reach a correct conclusion as to the cause of that type of headache which accompanies or follows an attack of migrana ophthalmica (scotoma scintillans, hemianopia fugax, etc.).

In all these forms of cephalalgia, no discoloration or swelling of the

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papilla is present, and they have been enumerated here merely to aid in differential diagnosis during the period when the ophthalmoscopic evidences are not conclusive. The subjective symptoms which are almost invariably complained of during the incipient stage of choked disk are also absent,—namely, unsteadiness of gait, weakness of the lower extremities, vomiting (although this is present in Bright's disease), dizziness, disturbances of memory, etc. It is furthermore important to remember that the headache in cerebral disease is sometimes relieved by eating. A similar phenomenon may be witnessed now and then in glaucomatous headaches.

In retrobulbar neuritis (especially in lead intoxication), although the ophthalmoscopic picture is somewhat similar, marked and characteristic disturbances of the visual acuity and of the field of vision occur. On the other hand, it is safe to state that, as a rule, both the functions remain normal in incipient choked disk. The main exception to this rule is found in those rare cases in which, in consequence of an accumulation of fluid in the ventricles or by reason of a distended "recessus nervi optici," the optic tract becomes compressed, or in which syphilitic granulation-tissue produces proliferation and compression of the optic tract itself. Under such conditions, during the period of the development of choked disk, and sometimes even while the papilla is still normal in appearance, impairment and loss of vision, accompanied by considerable restriction of the field of vision and various hemianopic disturbances, may take place.

On account of the eminent importance of the differential diagnosis in incipient neuritic swelling, mention is also made of that comparatively rare condition in which the papilla in high degrees of hypermetropia presents a normal appearance, but projects one diopter more or less above the level of the adjacent retina. In such cases, the most pronounced convolution and tortuosity of the vessels may exist, the veins in particular being wound around the central artery. From the region of the physiological excavation, grayish white but sharply defined velvety and shining bands of congenital connective tissue are spread out and encircle the blood-vessel trunks and in part conceal them. The striation of the upper and lower margins of the papilla in the direction of the retina is also more pronounced in these cases. Notwithstanding all this, the effect is not that produced by the typical cloud-like haziness that obscures the discolored papilla and its borders in incipient choked disk.

A still rarer source of error might arise in certain forms of astigmatism, where the lower and upper borders of the papilla have a streak-like, ill-defined, and hazy appearance; the confusion may be increased if nervous hyperæmia of the papilla is present at the same time. The results obtained by cylindrical lenses, together with the history of amblyopia since childhood, will guard against mistakes in diagnosis. Besides this, the subsequent developments—progressive swelling and increasing discoloration of the papilla—serve to distinguish beginning atrophy from the anomalous condition referred to.

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Schmidt-Rimpler has called attention to the presence of venous pulsation as one of the manifestations in brain-disease.

As the neuritic inflammation progresses, the cloudiness along the borders of the papilla increases, becoming denser in proportion to the area involved. Finally the margins become totally obscured, while the papilla takes on its characteristic "woolly" look, with the central area a dull grayish red. The diameter of the disk is often apparently increased. In the inverted image the papilla is seen as a dusky red disk, while the surrounding area assumes a hazy appearance, with dusky white rays visible here and there. The veins are distended and dip down abruptly as they pass over the edge of the boggy papilla to the retina below. At this point the venous coils are darker and sometimes nearly black in color. The vessels throughout look hazy and seemingly interrupted in their course. Now and then pale red or dark-colored hemorrhages, radiating in outline as a rule, may be detected along the borders of the spotted papilla.

On digital pressure it is possible to recognize pulsation and lateral displacement of the veins that descend from the cupola-shaped papilla.

In the upright image it can be noticed that the apex of the swollen disk generally projects about three diopters above the level of the retina, the actual elevation at this point being calculated as equal to one diopter. The tissue of the papilla looks as if it consisted of cloudy agar, interspersed with numerous tortuous and small blood-vessels, in the neighborhood of which minute hemorrhages are often visible. Another striking feature is the presence of lustrous, spindle-shaped, radiating, but irregularly grouped spots and blotches in the substance of the papilla (varicose sclerotic degeneration of the optic nerve fibres). The veins and arteries upon the papilla, when not obscured, are accompanied by broad, discolored, whitish bands and stripes. In a few instances the veins and arteries are contracted in calibre towards the middle of the papilla,—that is, near the place where they enter the physiological excavation. It must be admitted, however, that this area is seldom visible in the ophthalmoscopic picture.

External to the papilla the vessels in the retina are somewhat dilated, a pathological condition which furnishes additional proof that the lamina cribrosa suffers actual compression in the papilla. The fact must not be overlooked, however, that sometimes, even under normal conditions, the blood-channels are apparently more contracted near the centre of the papilla than near its edge, a circumstance which is explained by the existence of the connective-tissue layer, which begins to cover the lumen of the blood-vessels even at the physiological excavation. In addition, the writer has noticed that in the early stages of the neuritic process the dilatation of the veins extends as far as the periphery of the retina. In two cases of developed choked disk ("tumor cerebri" and "cysticercus multiplex cutis et cerebri") that came under the writer's observation, the venous branches appeared like spiral coils, while small extravasations of blood could be seen in the immediate vicinity of the vessels. The small hemorrhages in these cases arose

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from anastomosing branches of the superior and inferior temporal veins in the neighborhood of the macula lutea.

CHOKED DISK AND ITS SEQUELÆ.

The swelling of the optic papilla to which the designation "choked disk" applies develops from the stage of intra-ocular neuritis described in the last section in about two weeks, or possibly earlier in cases of tubercular meningitis.

The degree of swelling in the majority of cases is pronounced, amounting to one millimetre, or even reaching from two to three millimetres (equivalent to from five to six diopters above the level of the retina by ophthalmoscopic measurement). The tumefaction, as a rule, is limited to the territory of the disk and its immediate neighborhood; in consequence of this the circumference of the papilla is increased. Upon the disk, along its margins as well as on the retina, hemorrhages are not frequently found, although it is true that shimmering, yellowish-white dots, similar to those met with in retinitis Brightii, but less pale and brilliant, are occasionally seen in the retina. In this connection it is important to emphasize that in isolated cases of Bright's disease engorgement and tumefaction of the papilla may take place and simulate very closely the swelling accompanying choked disk of cerebral origin. It is possible that the paleness of the arterial blood-vessels in papillitis Brightii gives a somewhat different appearance to the ophthalmoscopic picture, but the presence of this condition is hardly sufficient unless it is accompanied by the typical nephritic alterations of the retina in the form of the well-known stellate configuration around the macula lutea. In such cases it is therefore always necessary to search for other characteristic symptoms of chronic interstitial nephritis. Patients have come under the writer's observation who presented a healthy appearance and neither complained of headaches or digestive disturbances nor gave any history of palpitation and other stenocardiac or asthmatic evidences,—the usual manifestations of Bright's disease. It was not until the ophthalmoscope was resorted to and a urinalysis was made that the diagnosis was established.

At times the patient's statements that he has occasional attacks of obscuration of sight may prove misleading. It is commonly accepted that in the progress of cerebral tumors, particularly when the patient is excited or engaged in active exercise, walking, etc., obscuration of the entire field of vision may result and last for several seconds or minutes, restoration of the former visual acuity then taking place. It is evident that in such cases there are alterations in the cerebral circulation or disturbances affecting the central artery of the retina. It happens that in Bright's disease anæmic attacks of blindness, resembling the visual disturbances occurring in brain-lesions, are also met with, but they generally last longer than the obscuration in the latter cases.

Only the typical neuritic swelling that is limited to the papilla or to its

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immediate surroundings has thus far been considered. It may happen, however, in rare cases of brain-tumor or in lesions of the skull and orbit, that the swelling is not protuberant and high, like the crown of a toadstool, but that the swollen papilla becomes flattened. Under these circumstances, opaque cloudy exudates and numerous hemorrhages reach far into the territory of the retina to the extent of a full diameter of the papilla or even beyond this. This complication has been called "neuro-retinitis or papillo-retinitis." It was formerly assumed that this disturbance was met with only as the result of meningeal lesions, the inflammation being transmitted along the trunk of the optic nerve to the eye (descending neuro-retinitis). Since then it has been established that papillo-retinitis is an attendant symptom in cerebral disease as well; moreover, it has been proved in brain-tumor that a typical choked disk may exist in one eye while the fellow-eye shows evidences of papillo-retinitis. On the other hand, choked disk may occur as an expression of tubercular and syphilitic meningitis. Even here, however, it is not a question of a purely inflammatory process directly descending to the globe, for Uhthoff distinctly states that in choked disk due to syphilitic meningitis he found the orbital portion of the optic nerve normal.

Elschnig also classed papillo-retinitis as among the changes affecting the papilla in brain-tumors. In this expression of the neuritic process it is common to find maculated, faintly shining spots similar to those appearing in true retinitis Brightii.

Regarding the course of the diseases under consideration, typical choked disk and papillo-retinitis, so far as the ophthalmoscopic picture is concerned, may continue for many weeks, sometimes even for one or two years, without any marked alteration except the occasional appearance of some new hemorrhages or fresh isolated areas of degeneration. It is extremely rare to meet with instances in which the choked disk has developed in two or three weeks, the actual lesion in these cases being usually an expression of a transitory meningeal affection. It is also exceptional to see the swelling rapidly disappear while, on the other hand, the cerebral lesion steadily progresses.

In the second stage of choked disk the patient frequently consults the physician because a mist seems to appear before his eyes, although the sight may be only slightly impaired. In fact, the rule is that normal vision continues for a long time in choked disk, unless the macula lutea is the direct seat of hemorrhage or degeneration, or unless the nerve itself (the optic chiasm or tract) is directly involved.

In addition to the symptoms already mentioned, particularly the invariable history of headache, during the period now under consideration, other general manifestations of cerebral disturbances become more marked and frequent. Chief among these are apoplectiform or epileptiform seizures, loss of memory, disturbances of speech, polyuria, deafness, paresis or paralyses of the ocular muscles, sluggish pupillary reaction, inequality in size of the pupils, mydriasis, etc.

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A peculiar impression is often produced by the apparent unconcern and good humor that the afflicted patient, whose facial expression is usually dull and fixed, displays in the presence of such a formidable host of general symptoms. (Oppenheim.)

Another striking feature is that the field of vision remains normal for a long time unless compression of the nerve itself or of the optic tract or chiasm exists. Slight restriction of the visual field, for a long time affecting only color-limitations, is sometimes present, and involves in particular the upper and lower portions of the field. The extent of the blind spot is usually increased. Microscopically, the writer has established the presence of a marginal, incomplete, and sickle-shaped process of degeneration affecting the nerve-fibres in the orbital and canalicular portions of the optic nerve.

However striking the long preservation of sight and of the visual field may be in the second stage of choked disk, equally so is the sudden and rapid impairment of both of these functions in the third stage of choked disk, or the stage of beginning neuritic atrophy. During this period the ophthalmoscope shows that the boggy papilla is growing paler and that the hazy exudations are becoming less opaque. When studied by the inverted image we may again begin to recognize the boundary lines of the papilla, particularly towards the side of the macula lutea. The vessels undergo contraction in calibre, especially the arteries. The venous loops in the retina become less numerous. White streaks are seen to follow the trunks of the chief blood-vessels. The hemorrhages have disappeared. The former markings on the lamina cribrosa are no longer visible. In the locality of the border of the papilla and around its circumference the chorioid discloses yellowish-whitish discoloration, and the pigment is irregularly piled up in this place. By means of this latter sign, as well as on account of the concealment of the stippled lamina cribrosa, together with the contraction and the attenuated appearance of the blood-vessels, the conclusion can be reached that a neuritic process or papillitis existed here and led to atrophy of the optic papilla.

If death does not occur during the first or, more frequently, during the second stage of choked disk, vision becomes reduced to the counting of fingers, and the field of vision is contracted or whole segments are obliterated. Color-perception for green is first lost; after this red and blue suffer contraction and consequent destruction; lastly, perception for white is lost, and finally total blindness results. A case of extraordinary rarity is reported by Gowers. A boy, twelve years old, as the result of a cerebral lesion, had atrophy of the optic nerves, followed by total loss of vision. Several years later he developed a new and distinct papillitis of the atrophied disks of both eyes, and this fresh disturbance was accompanied by the usual manifestations of intra-cranial tumor.

An exception to the usually grave prognosis is found in choked disk in the second stage as an expression of a syphilitic gumma of the brain-

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membranes. (It is very rare to find gummatous diseases of the papilla alone.) Under proper antisyphilitic treatment the choked disks in these cases often disappear and the vision may become normal again or nearly so. In certain rare cases (Uhthoff) a recurrence of the choked disk as well as a return of the general symptoms occurs. It is true that in these instances it might be assumed that the first attack was of syphilitic origin and was relieved by the proper medication (Case IV.), while the origin of the second choked disk could probably be traced to those rare expressions of Bright's disease in which neuritic swelling of the optic nerve takes place. In fact, in the case referred to, on the subsequent death of the patient the presence of chronic interstitial nephritis was established.

The prognosis is likewise not uniformly fatal as to sight in choked disk associated with malformations of the bones of the cranium. In the steeple-like and boat-shaped skull-deformities, as well as in hydrocephalus, the papilla presents a waxy, œdematous, and opaque appearance, with few or no hemorrhages. Even when the disk shows atrophic changes and contraction of its boundaries, a part of the visual acuity and field of vision may be preserved. In such cases the ophthalmoscope often reveals a pale rose-colored area along the nasal side of the papilla.

It is evident from the preceding consideration that brain-tumors and especially neoplasms occupying the posterior fossa constitute the most common cause of choked disk. New growths of the cerebellum and the adjacent locality produce engorgement of the papilla early in the history of the case. We are almost justified in concluding that those rare cases of brain-tumor, especially in the anterior hemispheres, in which no choked disk manifests itself, end in death before the neuritic swelling in question is able to develop. As an additional aid in diagnosis it is well to remember that the symptoms as a whole become steadily but surely and progressively worse, without any temporary objective improvement. In cases of syphilis of the brain-membranes, on the contrary (whether a gumma exists or not), the symptoms pass through a regular ebb and flow, temporary improvement alternating with sudden recurrences. (Uhthoff.)

In this connection reference must be made to another very rare condition associated with choked disk, or, more correctly speaking, with neuroretinitis (unilateral) during an attack of hemorrhagic pachymeningitis. In these cases, striking remissions long in duration and misleading in character make their appearance, so that the patient often considers himself almost well in mind and in body.

Further causes for choked disk are tubercular meningitis (with or without solitary tubercles in the brain), cysticerci, simple cysts, acute poliomyelitis (Wernicke), and cerebral abscess.

The cases of choked disk reported as having occurred in anæmia and chlorosis deserve particular mention. In the cases described by Gowers it is a question whether there did not exist a secondary anæmia associated, for example, with latent tuberculosis, since solitary tubercles may

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have been present without exciting any special disturbance. In such instances it would be possible to meet with choked disk even when no lethal result occurs, since spontaneous cure of the tubercles may take place. That this happens in other parts of the body has often been demonstrated, for instance, in tubercular areas of the lungs. Another strong doubt arises in reference to Gowers's cases when it is considered, on the one hand, how very rarely "chlorotic" choked disk has been reported, and, on the other hand, how often chlorosis itself is encountered by the general physician as well as by the ophthalmologist. The writer has observed choked disk in a girl fifteen years old, in whom the early symptoms were headache and the usual manifestations of chlorosis. After a duration of several months the choked disks were followed by beginning neuritic atrophy. Within two years the patient died from general tuberculosis with brain-symptoms.

Choked disk (or its incipient stage under the guise of intra-ocular neuritis) generally affects both eyes, but it is usual to meet with fully developed choked disk on one side while incipient choked disk or papillitis or papillo-retinitis is present in the other eye. In the majority of cases it seems that choked disk appears earlier on the side where the brain-lesion is situated. This rule is especially applicable to those neoplasms which force their entrance into the cavity of the brain or the orbit by way of the sphenoidal bone.

At times, as has been stated, choked disk arises as a result of cysticercus, cysts, cerebral abscess, and suppurative, traumatic, otic, and metastatic meningeal affections. Among the orbital causes for choked disk and papillo-retinitis (unilateral in such cases) mention must be made of tumors occupying the orbital walls, the lacrymal glands, the periorbita, and the optic nerve and its sheaths, as well as orbital leukæmia and caseous periostitis. It may likewise happen that retrobulbar neuritis may give rise to a swelling of the papilla closely resembling choked disk, together with extravasations of blood into the optic nerve and its sheaths at or near the locality of entrance for the central blood-vessels of the retina.

In conclusion it may be mentioned that the very uncommon neoplasms that start from the papilla or from the trunk of the optic nerve behind the sclera and by means of this path force their way into the eye present an ophthalmoscopic picture entirely different from that found in choked disk, so that an error in diagnosis is hardly possible. This also holds good for the cystoid new formations of the papilla.

PATHOLOGY AND MORBID ANATOMY.

A. *Pathological Appearances in the First and Second Stages.*—In a case of glioma of the pons in which general symptoms had persisted for months, Schmidt-Rimpler observed the following pathological changes accompanying the development of choked disk. In the lower half of the retina of both eyes, the portion immediately adjoining the papilla appeared slightly discolored; a similar although less marked condition affected the papilla

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itself. In the right eye, a vein coming from below attracted attention by presenting a distinct pulsation that could be traced over rather an extended area (even beyond the papilla),—a phenomenon that was not present before this. All these manifestations, however, so slightly exceeded the bounds of physiological conditions that a diagnosis sufficiently certain for pathological purposes was possible only by comparison with the previous state of the fundus. Section was made after two days. The retina in the neighborhood of the papilla appeared slightly discolored. The retinal vessels showed rather marked engorgement, but no distinct swelling of the disk could be detected. Near the globe the external sheath was found moderately dilated and reduced in resistance. Microscopically (Müller: paraffin), the bundles of nerve-fibres were found separated from the connective-tissue septa by broader interspaces, the septa being in part empty and in part filled with a translucent mass breaking up into small particles. These changes extended back behind the sclera as far as the point at which the central vein of the retina passes into the trunk of the optic nerve; beyond this the pathological dilatation disappeared. Close to the eyeball the cavity intervening between the dural and pial sheaths seemed broad. A multiplication of cell-nuclei, either in the sheaths or in the papilla and optic nerve proper, could not be discovered. In the papilla, little gaps could be seen between the nerve-fibres, the latter having been pressed apart. A rather large number of nerve-fibres gave evidence of the characteristic ganglionic thickening. In the retina some distended vessels could be seen, and this dilatation was particularly noticeable in certain single capillaries that ran across the fibre-layer in an almost vertical direction towards the ganglion-layer. The thickness of the nerve-fibre layer at the chorioidal ring was 0.41 millimetre. "It is evident that we have to deal here with an œdema of the papilla involving the nerve as far as the entrance-point of the central vein behind the sclera."

After noticing with the ophthalmoscope the beginning of papillo-neuritis, Ulrich within two weeks in two cases was able to make the microscopic examination. The ophthalmoscopic picture disclosed blurring of the borders of the papilla, slight prominence, and partial concealment of the blood-vessels, together with the appearance of ribbon-like hemorrhages aggregated around the papilla. Microscopic examination (Müller: celloidin) failed to show an intervaginal space of any consequence. The thickness of the nerve-fibre layer was 0.8 millimetre at the chorioidal ring. The disk was moderately swollen, and there was marked hyperæmia of the veins and capillaries. The bundles of nerve-fibres were pressed apart by an aggregation of fine granules as well as by small, hyaline, drop-like globules, and by larger, nearly round new formations with smooth granular contents. External to the papilla masses of hemorrhages were found in part in the nerve-fibre layer or upon its surface, and in part in the inner and the outer granular layer,—that is, in the subgranular layer. Others had broken through the layers in order to spread out upon the outer side of the

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layer of rods and cones. These extravasations extended into the retina for a distance equal to twice the breadth of the papilla. The nerve-fibre layer bulged out over the edge of the chorioid and pushed to one side the outer layers of the retina. In the neighborhood of the smaller veins and invading the vessels themselves small colonies of leucocytes appeared. Here and there a heaping up of blood-vessels occurred, while a nest-like hypertrophy of the nerve-fibres manifested itself, especially near the surface and next to the boundaries of the papilla. None of the alterations enumerated thus far passed beyond the limits of the lamina cribrosa centrally. In the optic nerve proper the bundles of nerve-fibres were separated from the connective-tissue septa of the supporting framework by strikingly wide interspaces. The central blood-vessels of the optic nerve trunk were compressed and empty.

In a third case of choked disk, which extended over a period of seven months, the same author found pathological evidences similar to those exhibited in the two preceding cases, with the addition of hydrops of the optic nerve sheath standing out from the pial sheath to the extent of three-fourths of a millimetre. The intervaginal supporting tissue was pushed out of place towards the dural sheath. Attaching itself between and upon the trabeculæ was found a granular mass, consisting of granules, globules, and numerous lymph-corpuscles, and containing at times larger cells, which either showed a nucleus with smooth, granular protoplasm or displayed signet-ring shapes interspersed with amylaceous corpuscles. In addition, Ulrich describes (1) œdema around the bundles of nerve-fibres, together with a marked increase of myelin, (2) œdema of the connective-tissue framework, and (3) œdema of the finer connective tissue in the nerve-fibres themselves.

In the last case, which was of longer duration, it is evident that Ulrich describes the existence of numerous cellular elements, even if he does not designate them as such. In the same way Deutschmann and Elschnig have reported the results of microscopic examinations in a number of older cases of choked disk in cerebral tumors; both found, in addition to unusually prominent œdema, frequent multiplication of cellular tissue between the nerve-fibres and behind the papilla.

Elschnig has examined twenty-eight choked disks in cases of brain-tumors. Since neuritic swelling was present at the first inspection in every one of these cases, it is impossible to determine how long this condition had existed before the ophthalmoscope was resorted to: they must be considered as older cases, however. Elschnig found the papilla projecting from one-half millimetre to one and two-hundredths millimetres above the level of the adjacent retina in consequence of "real œdema." In addition, he found the evidences of cellular infiltration in all the supporting and connective tissue of the papilla, as well as along the central blood-vessels down to their finest ramifications. The physiological excavation was filled with an œdematous new formation of connective tissue, with many blood-

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radicles, surrounded by an exudation of round and spindle-shaped cells, some of these being united by small strands of cellular connective tissue. The intermediary tissues—the retina and the chorioid—revealed evidences of inflammation in the neighborhood of the papilla. In the optic nerve perineuritis manifested itself, the process being most marked in the immediate vicinity of the lamina. Ampulla-like dilatation of the intra-vaginal space, with proliferation of its endothelial surface in certain sections, was visible. The arachnoid sheath as a continuous membrane, consisting of numerous strands of new connective tissue, was frequently found intimately adherent to the dural sheath. Gray degeneration of the medullary nerve-fibres with exudative swelling and breaking up of the medullary sheath into small drops and granules, and tumefaction of the axis-cylinder, were among the other prominent pathological signs in evidence. The central nerve-bundles of the optic nerve seemed least subject to degenerative changes. Here and there some of the scleral lamellæ that form the lamina cribrosa presented a convex protuberance on their ocular side. The central blood-vessels were normal and not compressed.

B. *Pathological Appearances in the Third Stage* (*Beginning Neuritic Atrophy*). In a case of two months' duration Ulrich found the papilla projecting 0.48 millimetre above the level of the retina. The tissue of the disk presented the appearance of a wide-meshed reticulum, in which isolated areas of sclerotic nerve-fibres as well as hemorrhagic effusions were visible. In the optic nerve itself (posterior to the lamina cribrosa) the marginal bundles of nerve-fibres were found disintegrated and replaced by hyaline débris which was deep black in color. Sub-pial œdema existed.

Elschnig says, "Eventually the papilla is converted into a network of connective tissue, which is densely fibrous even at this stage, but is still marked by great multiplication of cell-nuclei and round cells. This new formation cannot always be distinctly separated from the similarly hypertrophied tissue of the lamina cribrosa. As the chronic inflammatory process increases, thickening of the vessel-walls follows. This in turn leads to contraction of the lumen of the larger vessels and partial or total obliteration of the smaller blood-channels (arteritis and phlebitis proliferans)."

The final result is complete atrophy of the nerve-fibres, with development of connective tissue. The latter, when seen in the ophthalmoscopic mirror, gives off a white reflex and conceals the stippled markings of the lamina cribrosa. The bulk of the papilla becomes diminished, with the result that the retina is drawn in towards the centre of the papilla, so that the connective-tissue new formation developed at the seat of the original tumefaction, in consequence of the close intergrowth with the migrated pigment-epithelium, remains fixed in position. On the other hand, the tissue of the retina is capable of being retracted over this point. This explains the origin of the "neuritic cone" in atrophy after choked disk (Elschnig).

Choked disk as an accompaniment of cerebral syphilis has been met

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with in fourteen per cent. of cases (Uhthoff),—in eight per cent. as a result of gummatous basilar meningitis, and in six per cent. as a consequence of true syphilitic cerebral lesions.

Microscopic examination of the swollen papilla reveals a pathological condition identical with that in cerebral tumors in general, as in the cases described by Elschnig. In the optic nerve proper, behind the sclera, no inflammatory alterations exist, these changes being limited to the perineurium,—*i.e.*, in the intervaginal spaces. The perineuritic process leads to such cell-proliferation and new formation of granulation-tissue, especially in the anterior division of the intra-vaginal space, that the outer nerve-sheath becomes separated from the inner sheath by a coarse partition of proliferating, highly cellular granulation-tissue, thus obliterating the ampulla-like dilatation of the intra-vaginal space. This new-formed and proliferating connective-tissue septum, now and then containing chalk deposits, extends ordinarily along the inner side of the optic nerve and generally more posteriorly. In the most posterior and orbital portion of the optic nerve, however, no pronounced pathological signs affecting either side can be pointed out.

It follows, therefore, that the choked disk in these cases cannot have a "descending" origin,—that is, the inflammation is not directly transmitted from the orbital cavity to the optic nerve and its sheaths. A descending process is likewise excluded in the cases reported by Elschnig, since in eight of twenty-one cases in which the optic nerve was examined through its whole course it was found normal in its orbital portion and without inflammatory changes in the perineurium and in the septa, the seat of the inflammation being limited to the papilla, to the portion of the nerve behind the sclera, and to the part occupying the bony canal,—in other words, to the more vascular areas of the optic nerve, where, as a consequence, according to Elschnig, the inflammatory proliferation would be more pronounced.

THEORIES AS TO THE ORIGIN OF CHOKED DISK.

According to Türck and von Graefe, increased intra-cranial pressure by compressing the cavernous sinus produces stasis in the inferior and superior ophthalmic veins which empty into the sinus. The stasis which then ensues in the central vein of the retina leads to engorgement and œdema of the optic papilla, especially if the compression in the first place causes œdema of the connective tissue of the lamina cribrosa, by which means the outflow of the venous blood is still further obstructed.

Sesemann (1869) and later Merkel demonstrated that the superior orbital vein, into which the central vein of the retina flows, communicates with the veins of the face by means of the angular vein above the inner commissure of the eyelids. By this anastomosis collateral circulation could be easily established, so that any stasis produced by compression of the cavernous sinus would be promptly relieved.

Parinaud believes that just as an elevation of the pressure within the

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cranial cavity leads to hydrops ventriculi and dropsy of the brain, in the same manner a dropsical condition of the optic nerve with consequent engorgement might originate.

Ulrich, in like manner, attributes the œdema to the optic nerve alone, the nerve compressing the central vessels (the artery and vein) in their axial course.[1]

Schmidt-Rimpler (1869) and Manz (mechanical theory of transmission) showed that, on account of the increased pressure, some of the cerebral fluid is squeezed out of the cranial cavity and forces its way into the intervaginal cavity of the optic nerve (the space discovered by Schwalbe). This accumulation dilates still more the ampulla-like inflation behind the sclera which is normally present. Gradually some of the lymph escapes into the trunk of the optic nerve itself, œdema of the lamina cribrosa follows, and, by virtue of this, compression of the central blood-channels, with consequent tumefaction of the papilla, results. The engorgement that now arises in the smaller and much distended blood-vessels leads to the production of lymph and serous exudation in the neighborhood of the vessels. The microscopic foundation for this theory was based on the observations made by Parinaud, Ulrich, and Schmidt in cases where ophthalmoscopic investigation had established the initial existence of choked disk. These cases furnished the anatomical basis for the compression of the central vessels of the optic nerve trunk by means of the primary œdema. Uhthoff makes the same observation in the case reported by him.

Later, Leber expressed the opinion that in cerebral tumors œdema of the papilla and of the nerve was not directly produced by the heightened intra-cranial pressure,—in other words, that the œdema was not primary, but that certain phlogogenic substances furnished the exciting cause, and by forcing their passage into the nerve and to the papilla, there excited primary inflammation with secondary tumefaction of the inflamed tissues. The main argument adduced is the evidence supplied by the microscope that in every case examined by Deutschmann and Elschnig, after the announcement of Leber's views, not only marked œdema but also chronic inflammatory multiplication of the cell-nuclei manifested itself, not merely in the immediate territory of the vessels themselves, but even in the central connective tissue as well as the septa in general,—a pathological process which neither Schmidt-Rimpler nor Ulrich noticed, although they described pronounced œdema between the bundles of nerve-fibres.

In explanation of this it may be well to remember that although Ulrich failed to find an increase of cellular elements in the two cases in which he had seen the papilla in a normal condition a short time before, as in Schmidt-Rimpler's cases, nevertheless in the third case, which was of longer duration, he describes numerous cellular elements as then existing in the perineural tissues. Even Elschnig states that in a few instances in which it was impossible to detect anything except cloudiness and distention of the veins of the papilla in cases of meningitis, he was also unable to discover micro-

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scopically that inflammatory proliferation which he almost invariably found in the many cases of fully developed choked disk observed by him. These cases, however, were older and had persisted for a much longer time than those described by Ulrich and Schmidt-Rimpler. These phlogogenous factors, whose existence still remains unproved, are likewise supposed to produce the discoloration that shows itself in the membranes of the brain near the seat of the tumors, an area in which microscopic examination has revealed the same cell-proliferation.

This controversy regarding the initial stage of choked disk, turning on the question whether or not the inflammatory changes followed by secondary œdema are primary, could be readily decided if it were possible to prove the existence of these specific factors of inflammation in the cerebro-spinal fluid in cases of tumors of the brain,—in fact, all lesions that produce choked disk. To do this it would only be necessary to collect some of the fluid after section and determine by experimental use whether it would give rise to the same inflammatory changes that we meet with in choked disk. Inasmuch as this proof is not forthcoming, no strictly scientific objection can be raised against the microscopic results in favor of the purely primary origin of the œdema in question obtained by Ulrich, Schmidt-Rimpler, and others.

In the mean while the following deductions suggest themselves: The ophthalmoscopic finding, that in incipient or in developed choked disk the papilla is respectively more opaque or more transparent, cannot be used as an argument either for or against the question at issue, since the degrees of translucency vary; for instance, it is well known that in hydrocephalus (and especially in peaked and boat-shaped skull-deformities), as well as in retrobulbar neuritis, the swollen papilla has a far more translucent look (somewhat like the appearance of congealed white vaseline) than in the majority of choked disks in brain cases. Again, even in brain cases the disk may sometimes be found strikingly transparent, this condition depending on the intensity of the œdema. Under all circumstances, however, it cannot be decided by the ophthalmoscopic picture whether the œdema is primary or inflammatory.

Even should it be assumed that the phlogogenic substances in question furnish the exciting cause for choked disk, it would be necessary to predicate their existence in all the lesions that lead to choked disk, including such a variety of disturbances as tumors of the brain, periosteal growths, as well as growths springing from the surrounding bones, cysticercus, benign and malignant neoplasms, aneurisms of the carotid and of the ophthalmic artery, cavernous and orbital new formations, pachymeningitis, hydrocephalus, meningeal affections, superior polioencephalitis (hemorrhagic), retrobulbar neuritis, leukæmia, etc.

Why do not intra-ocular tumors of a nature similar to the neoplasms found in the brain give rise in a similar manner to choked disk, especially since in the former the phlogogenous factors could reach the papilla by a much shorter and more direct path? Why is choked disk not found as an

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invariable expression of retrobulbar neuritis? Why in this disturbance, even under the most favorable circumstances, is hardly ever anything more than optic neuritis found, when it is incontrovertible that retrobulbar neuritis involves the papilla, that it is of long duration, and that it is a disease which shows (as microscopic examination clearly demonstrates) the most pronounced proliferation of the connective tissue of the optic nerve (much more marked than that which occurs in the cerebral lesions)?

Is the multiplication of cell-nuclei so very marked when compared with the numerous nuclei that are present even in the normal and healthy condition of the optic nerve?

All these considerations show the urgent demand for discovering, if possible, some distinctive mark and common cause for all choked disks, whether of cerebral or of orbital origin.

It can be taken almost for granted that in pointing out the main features of the ophthalmoscopic picture of choked disk every examiner will state that he finds some particular vein or both central veins distended. It is equally evident that if compression produces this dilatation the pressure exerted cannot be great, because the appearance presented here is totally at variance with the picture of pronounced compression with which all are so familiar,—for instance, in thrombosis of the central vein of the retina. It is therefore justifiable to assume that a comparatively slight obstacle impedes the outflow of blood in the central vein. Even under such circumstances, however, it is evident that the egress of blood from the smaller venous branches upon the papilla will also be interfered with, and it follows as a natural consequence that the serous exudation as well as the resulting discoloration will be most pronounced in those places where the nerves as well as the small (and in the present case) distended blood-vessels are most numerous,—that is, in the upper and lower portions of the nasal half of the optic disk. This supposition is verified by the prominence and discoloration which actually occur in the locality indicated.

How is this compression of the central venous trunks of the retinal vessels produced? It is plain that compression can be caused by anything that interferes with the normal flow of blood in the central vein, during its course along the axis of the optic nerve, in its oblique passage from the nerve-trunk into the intervaginal space, in its exit through the dural sheath, and, lastly, during its progress and distribution external to the sheaths.

These preliminary facts being admitted, according to the writer's researches the origin of the choked disk takes place in the following way:

The vein passes out from the nerve-trunk through a narrow slit which can often be made out even by the unaided eye as a gray line in the pial sheath. (As a rule, the vessels will be found close together here, the opening for the central vein being a little nearer to the eye than the point of entrance for the central artery of the retina.) If the vein is compressed in the constricted orifice mentioned above (whose lip-like edges swell under

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the slightest œdema), the slight initial stasis that often occurs can be explained. The œdema of the optic nerve arises as a result of the increased tension and the consequent obstruction offered to the outflow of blood and lymph from the cranial cavity, the pressure in question being looked upon by the pathologist as a common and almost constant condition and recognized by the clinician in the living subject as well (the so-called "tense dura"). This compression of the central vein in the passage described above has been demonstrated by the writer in brain-tumors by a series of microscopic sections. It is evident that after hardening and preserving the specimen it is not possible to detect the œdema unless it assumes enormous proportions. It is also evident that this compression and stasis tend to increase the existing œdema. As a consequence, dropsy of the cavity between the sheaths of the optic nerve may develop, so that the intervaginal space frequently presents an ampulla-like dilatation in choked disk, and is found filled with fluid. This accumulated fluid, under increased pressure, gradually encroaches upon the external sheath and constricts its contents so that the central vein as it passes obliquely from before backward through the intervaginal space is put on the stretch and suffers compression at the inner surface of the dura. In the few instances of cerebral disease (especially in cases of brain-tumor) where this ampulla-like inflation is found unassociated with choked disk, we may assume that death resulted before sufficient compression of the venous channels took place; a condition which, in individual cases, often depends on anatomical causes that affect the exit of the vein from the nerve or from the external sheath.

Figs. 1, 2, 3, 4, and 5 represent a series of cross-sections of the optic nerve in a case of brain-tumor and choked disk of long standing. The sections extend from a point directly behind the entrance of the central vessels in the pial sheath to their place of exit from the dural sheath.

The writer has proved by microscopic sections (Figs. 1, 2, 3, 4, and 5) that the central vein (which makes an abrupt turn in its passage through the external sheath) undergoes constriction and compression by reason of the dilatation of the ampulla-shaped space,—*i.e.*, in consequence of the fact that the external sheath is forcibly pressed out and rendered tense by the accumulated fluid and constantly increasing dropsy of the intervaginal cavity, so that the vessel incarcerated in the wall of the external sheath can receive only the most limited blood-supply, and is often found greatly reduced in calibre. It is obvious that the constriction that takes place may vary in degree and extent (Figs. 2 and 3), although in the incipient stages it would be hard to establish this by microscopic examination. External to the constricted areas, at the minute opening in the optic nerve trunk and in the dural sheath, the central vein may show varicose dilatation in certain parts (upon the papilla, in the axis of the optic nerve, and in the intervaginal space). (Fig. 2.) This condition can be readily explained, particularly if it is remembered that both before the constriction is reached (Fig. 2), as

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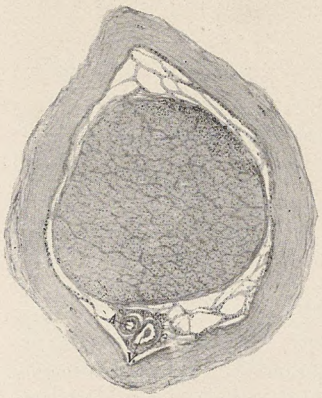
FIG. 1.

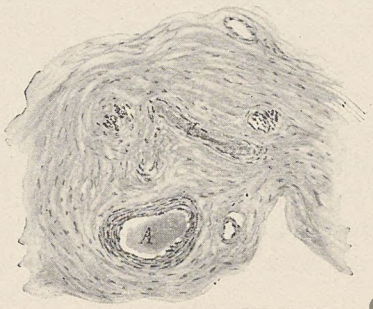


Central vessels immediately after their passage through the pial sheath (low power). *A*, central artery of the retina; *V*, central vein of the retina.

FIG. 2.

Central vessels of Fig. 1 under higher power. *A*, central artery of the retina; *V*, central vein of the retina; *O*, periphery of optic nerve; *D*, inner layers of dural sheath.

FIG. 3.

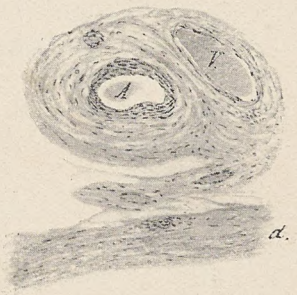


Central vessels in wall of external sheath.

FIG. 4.

Central vessels in the outer layers of the dural sheath. *A*, central artery of the retina; *V*, central vein of the retina, with hemorrhage.

FIG. 5.



Central vessels external to the dural sheath. *A*, central artery of the retina; *V*, central vein of the retina; *d*, outer layers of the dural sheath.

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well as after the point of incarceration is passed (Fig. 5), this blood-vessel receives the contents of a number of venous branches; out of these the blood pours into the unobstructed portions of the central vein, and dilatation follows. (Figs. 3 and 4.) In the intervaginal space the central vein derives its most abundant blood-supply from the pial branches of the nerve-surface; external to the sheaths the external dural branches, which are very numerous and profuse, may distend the vein in its central course behind the compressed and half-drained section of the vessel in the dural wall. (Fig. 4.)

In exceptionally rare cases of choked disk no mention is made of ampulla-like inflation of the intervaginal space. Since it has not been determined what degree and extent of dilatation should be considered pathological, it is possible that a moderate dilatation may have remained unnoticed; or the ampulla may have become obliterated by perineuritic proliferation. This obliteration may have had an effect on the central vein similar to that produced by compression, so that it is possible in certain cases of choked disk to find the ampulla absent. In fact, in order to bring about constriction of the dural portion of the central vein a pathological process need only affect the medial lower quadrant of the sheath-cavity of Schwalbe, as, for example, even an incipient hydrops of the intervaginal space. As a rule, however, in choked disk this ampulla remains visible, appearing tense and distended on microscopic examination, although it will not be found necessary to tie the moist and glistening nerve in its orbital portion, since only a part of the fluid that fills out the ampulla escapes. In children, and occasionally in adults, the external sheath is found strikingly thin, a condition the existence of which must also be taken into consideration in certain cases.

In addition to the causes enumerated, compression of the vein as it leaves the nerve may be brought on by leukæmic infiltration and by tumors of the nerve-sheaths, etc. It is a recognized fact that neoplasms of the nerve-sheaths generally do not give rise to choked disk until late in their development, for it is only after a gradual growth, lasting for years, that they extend forward and follow the path taken by the blood-vessels in their exit from the nerve and its sheaths. Blindness or marked disturbance of vision occurs much earlier. The time at which this extension forward takes place varies, and we may safely assume that this variation is directly dependent in a given case on the exact position in the optic nerve of the place of exit of the central vein and the point of entrance of the artery. We know that in some instances this has taken place as close as six millimetres behind the globe; in others, the distance has been found to equal seventeen millimetres. On similar grounds the variable œdema of the ampulla can be accounted for. Unquestionably the result will differ considerably if the outflow of venous blood and the entrance of arterial blood respectively are obstructed, on the one hand, almost immediately behind the globe, or, on the other hand, not before a distance of from fifteen to seven-

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teen millimetres is reached. In fact, the origin, development, and general picture of choked disk will vary accordingly.

In addition, the result of the writer's investigations, based on comparative anatomy and embryology, and verified on the cadaver in thirty cases, must be taken into account. These researches show that the central vessels enter the nerve in the lower nasal quadrant.[1] In other words, applying this rule to the present case, it follows that, no matter what the cause may be that produces compression, no constriction of the vessels in their passage from the nerve, no œdema due to arrest of circulation in the papilla, and consequently no choked disk, will arise so long as the pressure is confined to the temporal side of the nerve. This happens, as a rule, in retrobulbar neuritis in which the inflammation is limited to the temporal, maculo-papillary (central scotoma) bundles; that is, to the side of the optic nerve turned away from the point of entrance of the vessels. This is the usual course in retrobulbar inflammations, in spite of the fact that the affected portions in this form of neuritis show a much more marked multiplication of cell-nuclei than in choked disk. If, on the contrary, the neuritis involves the whole cross-section of the nerve, producing amaurosis, or extends at least to the threshold of the vessels, choked disk results, a condition met with in grave neuritic lesions. For this reason Uhthoff describes a choked disk accompanying a perineuritis (*lues cerebralis*) in which the process began at the globe and extended backward along the inner side of the optic nerve.

In this connection we must also consider the so-called inflammatory multiplication of nuclei, which, according to the theory of the phlogogenic origin of choked disk, is looked upon as a primary manifestation. In the first place, the writer's investigations warrant him in stating that the increase of nuclei is not so strikingly large, especially in view of the numerous nuclei and nucleoli that are normally present in the optic nerve. But, even if it is admitted that a slight multiplication of nuclei manifests itself in cases of long-continued choked disk, this process is apt to be looked upon as a secondary expression and a necessary sequel of stasis of long standing, allied to the analogous multiplication of cell-nuclei met with in the kidneys and liver, in which there is obstructed circulation resulting from cardiac lesions (*cirrhosis cardiaca*). In fact, on anatomical examination it will be found that the masses of nuclei are heaped around small and distended veins, and are especially prominent on the surface of the optic nerve, in the pial and arachnoid sheaths. In incipient choked disk, as has been stated, neither Ulrich nor Schmidt noticed this multiplication of nuclei, a proof that this condition is not essential to the formation of choked disk. On the other hand, the necessary factor in the latter disturbance is the œdematous swelling. This, it is obvious, must be due to another cause; in spite of the fact that Elschnig expressly states[2] that it may be impossible to find choked disk even though the multiplication of nuclei is established.

[1] Deyl, Anatomische Anzeiger, Jena, 1896.

[2] Loco citato, pp. 242, 248, 249, 265, 275, 290, 292.

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In brief, the œdema of the papilla, which stands in direct causal relation to the swelling, does not depend upon the multiplication of nuclei, but is due to some interference with the circulation of blood in the central vein. This obstruction is produced in various ways, the most common cause being increased intra-cranial pressure. As a result of this condition the outflow of blood and lymph from the optic nerve is impeded. This in turn leads to primary œdema of the trunk of the optic nerve and its sheaths, dilatation of the intervaginal space, compression of the walls of the external sheath, and, as a final consequence, more or less constriction of the central vein as it passes on in its oblique course.

Jansen attributes the cause of choked disk in his cases of sinus-thrombosis to leptomeningitis of the optic nerve.

An incomplete thrombus of the central vein of the retina in its dural, intervaginal, or intra-neural course, or a hemorrhage in the regions referred to, may give rise to a similar obstruction of the venous circulation, with the production of a similar œdema; in this class must be placed the numerous and spontaneous thrombi-formations in high degrees of anæmia (chlorosis, excessive loss of blood, etc.).

Reference must likewise be made to the fact that the œdema which microscopic demonstration establishes in choked disk is far in excess of the inflammatory changes which both Deutschmann and Ulrich invariably describe as a chronic process. More than this, according to the admission of Elschnig, the œdema in question is much more marked during life than that found after the globe is preserved and hardened. In other words, it has been proved that papillæ whose prominence, estimated by the ophthalmoscope, ought to have been much higher, were found after death to be much less elevated. For example, in one of Elschnig's cases, after the globe was hardened, measurement of various parts of the papilla showed that the pathological elevation amounted to from 0.50 to 1.20 millimetres, equivalent to from one and one-half to three diopters by ophthalmoscopic examination, while, as a matter of fact, during life the prominence always reached from three to five diopters or more. From this it can be seen that the œdema must have decreased after hardening of the nerve, so that it is not always possible or necessary to demonstrate by strict microscopic proof a constriction of the vessels at their place of entrance into the nerve, even though this condition existed during life.

In conclusion, it is evident that the course of the central blood-vessels and the variable distance from the globe at which they enter the optic nerve, together with the rapidity and the intensity of the compression, tend to explain why certain cases of brain-tumor, etc., give rise to a papillo-retinitis instead of the typical choked disk, their usual manifestation.

Deutschmann experimented on rabbits by injecting agar into the sheaths of the optic nerve and the cranial cavity. Immediate venous dilatation and swelling of the papilla followed. If the increase in pressure arising in this way could be made to last, as in tumors of the brain, we should be

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justified in expecting permanent swelling of the papilla, provided the disposition of the central vessels is the same in rabbits as in man.

The opinion of Albrecht von Graefe, that "papillo-retinitis" is a descending neuritis due to an inflammation of the lining membranes of the brain, is contradicted by the experience of every physician who makes use of the ophthalmoscope. Abundant evidence exists that this disturbance may also occur in tumors of the brain. Elschnig found that among the one hundred and eighty-one reported cases of brain-tumors in which a post-mortem section was made, twelve, or seven per cent., showed the typical picture of neuro-retinitis, like that met with in retinitis albuminurica (hemorrhages, and varicose degeneration of the nerve-fibres around the papilla, especially around the macula lutea).

Another proof that the phlogogenous substances referred to extend to the papilla is said to be furnished by certain cases of brain-tumors in which trepanation was followed by arrest and rapid disappearance of the choked disk. These cases, however, merely prove that operative interference produced a strikingly rapid reduction of the swelling. It is certainly not necessary to explain the so-called "cure" by assuming that the trepanation dislodges the factors of inflammation and forces them to retreat from the papilla. On the other hand, it is much more reasonable to conclude that with the subsidence of the intra-cranial pressure (Bruns) the compression of the vessels is relieved and the œdema vanishes as a natural consequence. At all events, it is hard to understand what bearing the operation and its results would have on the question whether the œdema or the inflammatory process is the primary factor in the production of choked disk.

THE TREATMENT OF CHOKED DISK.

In the large majority of cases of choked disk, as has been previously stated, the visual acuity and the field of vision remain nearly normal and undisturbed for a long time. This being the case, it is self-evident that if the original disease which furnished the exciting cause for the production of choked disk could be removed, a favorable prognosis would be justifiable. In fact, the beginnings of the practical demonstration of this deduction can be found even at present in the surgical interference resorted to in tumors of the brain, cases being reported by Horsley, Gowers, Bruns, and Bergmann, in which it was possible to remove the cerebral growth. What is more, it was proved in these cases that the evidences of increased intra-cranial pressure, particularly headache and choked disk, subsided [1] (Zeller,

[1] In this connection the translator must refer to a case of his own in which a brain-tumor of large size was successfully removed, followed by a complete subsidence of the general symptoms, rapid disappearance of the typical choked disk, with no restoration of vision in the eye in which blindness existed before the operation, but sufficient restoration of sight to do ordinary work in the eye in which perception remained. The most remarkable thing about this case is that at a recent visit, almost four years after the operation, there had been no recurrence of the local growth nor the slightest return of any general or local symptoms.

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Berlin, 1895). In addition, it was established that marked general nervous symptoms ceased as soon as a trepanation was made, even without a resection of the tumor or of the cerebral tissue. It is true that in similar cases a return of the disturbances may at times be expected, and even the reappearance of choked disk in the atrophic optic nerve (de Schweinitz and Thomson). Amelioration of the general symptoms and of the choked disk has been also observed after puncture and allowing the accumulated cerebro-spinal fluid to escape from the spinal canal (Quincke's method).

De Wecker has suggested incision of the ampulla-shaped, dropsical dilatation of the nerve-sheaths behind the sclera. If a further advance in diagnosis and therapeutics in general would justify such an operation, its frequent execution could be thought of. If this should be the case, it need only be mentioned here that, for reasons previously stated, nothing more would be necessary than to cut out with the scissors a portion of the external sheath towards the outer and upper side that is removed from the place at which the central blood-vessels enter the sheath (the vessels being readily determined in these cases upon the external surface of the dural sheath).

In tumors of the brain the routine treatment is iodide of potassium. Even in non-syphilitic cases this remedy at times produces at least subjective relief.

In choked disk in which a syphilitic origin can be established, mercurial medication (preferably in the form of inunction) yields good results. It is necessary, however, to observe the usual precautions if albumen is found in the urine, or if disturbances of the digestive tract, ptyalism, etc., develop. If the patient presents himself in the stage of beginning atrophy, even if the papilla still remains swollen, it is safer to use iodide of potassium alone, since mercury merely hastens the reduction of vision that accompanies the atrophic changes.

A prognosis relatively far more favorable is furnished in the treatment of intra-ocular neuritis and choked disk in retrobulbar neuritis in the subacute or chronic form, and sometimes even in the acute variety.

A special group, whose existence can be based on clinical grounds at least, is formed by that variety of neuro-retinitis in which it is impossible to establish orbital or central tumors or nephritic disturbances of any kind. Knies saw this form of neuro-retinitis in intensely anæmic young women in whom the ocular symptoms, after persisting for months, finally yielded. This form of neuro-retinitis is generally met with in syphilitic affections of the optic nerve. To this class, perhaps, belong those rare instances of neuro-retinitis appearing in menstrual disorders, as well as the neuro-retinitis after scarlet fever, measles, diphtheria, typhoid fever, influenza, whooping-cough, and pneumonia.

In those forms of neuro-retinitis not due to tumors, the papilla is reddened and discolored, the discoloration extending over into the retina the diameter of the papilla or more. There is only slight swelling of the disk, however, and hemorrhages are absent, or small and few in number.

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The syphilitic group is important, and must be mentioned separately. Most frequently in middle life, within from one to four years after infection, dimness of vision, unaccompanied by pain, manifests itself; visual acuity is reduced to one-third, and concentric contraction of the field of vision reaches almost thirty degrees. Under antisyphilitic treatment, after a few months, generally complete, though in some instances only partial, restoration of visual acuity is obtained, and the visual field enlarges, although not to a normal degree. The papilla continues to show slight pallor, and its outlines remain, on the whole, rather sharply defined. The younger the patient and the earlier the ocular disturbance appears after the initial infection, the more favorable is the prognosis (Horstmann).

After great loss of blood the neurotic appearance of the papilla which sometimes develops, instead of the usual atrophy, merits special mention. (See Atrophy of the Papilla and of the Optic Nerve.) The cases of neuritic atrophy of the papilla in internal hydrocephalus of the young are also interesting. In these, associated with anosmia, a continual dropping of fluid, amounting sometimes to one pint daily, may take place from one or both nostrils. According to Leber and Dollense, this fluid in its chemical constituents corresponds to the cerebro-spinal fluid.

SUBDIVISION II.

RETROBULBAR NEURITIS.

This disease, which, in contradistinction to choked disk due to tumors and the forms of neuro-retinitis just considered, begins with disturbance of central vision, has been studied clinically chiefly by Graefe and Leber. This affection and the researches regarding its pathological anatomy (Samelsohn, Bunge, Nettleship, Uhthoff, Vossius, Sachs) have led to the discovery of the anatomical seat of those fibres which supply the macula lutea and therefore are in direct connection with central visual acuity. These fibres occupy the temporal side of the disk, and constitute nearly one-fourth of the area of the papilla. They form a wedge-shaped segment whose blunt apex reaches to the central blood-vessel trunks, while the base lies at the temporal edge. They have received the name of the papillo-macular bundle (Bunge). The fibres continue their wedge-shaped arrangement even behind the papilla, with the difference, however, that the apex lies in the axis of the nerve near the central vessels, and in this place, as a rule, other fibres become intermingled with them. At their entrance into the nerve the vessels are found surrounded by other fibres which do not belong to the papillo-macular bundle.

At a point in the orbit at some distance from the optic canal the base of the papillo-macular bundle can no longer be traced to the pial sheath; on the contrary, in this locality it will be found separated from the sheath by other fibres, and, while it is here placed somewhat temporally and occupies an eccentric relation to the axis of the nerve, it fails to reach the

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sheath, from which in the optic canal it is found still farther removed, somewhat towards the axis. Its intra-cranial section is more inclined and oval and lies below to the temporal side.

GENERAL PATHOLOGY.

The papillo-macular bundle can be distinguished from the rest of the nerve-fibres even on microscopic examination, this difference being especially apparent in the cross-section of the nerve. The temporal quadrant is of a different color, as well as denser; the trabeculæ cannot be seen, while normally in this portion can be made out at least the indication and outline of the septa that form the connective-tissue framework. If a staining process has been used, the section takes on a deeper stain than the healthy portion. At a later stage the periphery of the nerve may be found flattened in the affected region.

Microscopically an interstitial inflammation of the connective tissue of the septa can be recognized; the proliferating area looks less dense and firm, the septa are broader and show a greater number of round cells, generally with a single nucleus; besides this, particularly in chronic cases, spindle-shaped cells appear not merely in the septa but also in the nerve-fibre bundles, the process being evidently a proliferation of the glia elements. The engorgement with the new formation of blood-vessels in the septa is striking. In the neighborhood of these vessels a covering of round cells is often found, although it is exceptionally rare to meet with an actual piling up, a nest, as it were, of these cellular elements. At the point where the wedge-shaped base of the affected nerve-bundle is in contact with the pial sheath, this enveloping membrane likewise shows granular degeneration and distention of its vessels.

In prolonged and chronic cases atrophy of the bundles of nerve-fibres results, but in the majority of instances this is only partial. The proliferating and infiltrated tissue of the septa compresses the nerve-bundles, disintegration of most of the fibres follows, and a granular, fibrillar mass develops, in which here and there a few nerve-fibres can be found preserved, some still supplied with their medullary sheath, while in others merely the axis-cylinders remain. Rarely a complete disappearance of the nervous elements takes place in any one bundle, so that as a result the septum is empty, or filled only with a fibrillar, wool-like mass, or replaced by a nodular reticulum derived from the connective tissue of the septa. It has not been definitely decided whether the degenerative changes and hypertrophy of the walls of the septa are merely relative,—that is, a consequence of the lack of fibres,—or whether the reverse is true, the lack of fibres being a result of the inflammatory swelling of the septa. In the former case the multiplication of nuclei (these being crowded together into a smaller space) would be merely a relative increase due to the shrinking and collapse of the walls of the septa.

The most marked manifestations of the disease are found in the optic

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canal or in the anterior portion of the nerve behind the eye (Uhthoff), but it is always the temporal eccentric bundle which is affected, so that the designation "neuritis axialis" is applicable only to those cases in which the inflammation has extended to the axis of the nerve.

Sachs found the most pronounced atrophic alterations of the optic nerve fibres in the optic canal at the point where, under normal conditions, a rather large vein (the vena centralis postica) enters the affected papillo-macular region. He found this vessel surrounded by a dense, small-celled infiltration which involved even the smaller branches between the septa.

If this pathological picture is taken into consideration, together with the way in which the nerve-fibres pass over from the papilla into the retina, it is not difficult to infer the nature and degree of the disturbance of visual acuity and of the field of vision, as well as the general ophthalmoscopic condition, and the anamnestic statements of the patient can be readily understood.

In their exit from the papilla the optic fibres simply cross over without interlacing and without passing from the deeper into the more superficial layers; for this reason the fibres of the papillo-macular bundle that are nearest to the temporal side—that is, situated along the margin of the optic disk—terminate on the temporal side in the immediate vicinity of the papilla, while the nearer the fibres lie to the central blood-vessels the greater is the distance from the papilla that they have to travel before they are spread out in the macula lutea. If a pathological process affects the papillo-macular bundle, a defect in the visual field will show itself corresponding to the area of the retina that extends laterally from the temporal margin to beyond the fovea centralis. If it can be imagined that the axis of the optic nerve, by which the object fixed is connected with the image in the fovea, emanates from the fovea centralis, it can be understood that there will be more extensive implication of the nerve-fibres that lie in the retina to the nasal side of the fovea centralis as far as the papilla (or, as regards the visual field, to the temporal side of the point of fixation and extending to Mariotte's blind spot); a far smaller section of the retina would be involved in a temporal direction from the fovea centralis,—therefore, in the visual field, to the nasal side of the point of fixation. The fibres of the papillo-macular bundle, which ought to extend completely around to the macula, are forced to undergo a curvature as they pass from the temporal, wedge-shaped portion of the papilla, so that those fibres through the whole extent of the retina form a horizontal oval group whose smaller section is represented by the wedge-shaped area in the papilla, and whose larger portion is filled in by the nerve-fibres that are inserted somewhat temporally from the fovea centralis. On the contrary, in the visual field, the implicated portion—according to the law of the projection of retinal images, the broader, shorter part of the egg-shaped defect—will be situated somewhat nasally from the point of fixation, and the narrower, longer por-

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tion will extend nasally as far as Mariotte's blind spot, this blind spot increasing the extent of the defect on the temporal side. It must be remembered that this schematic arrangement holds good only in those cases in which the lesion is limited to the papillo-macular bundle.

The scotoma in the field of vision is a horizontal oval. It is central, and extends in typical cases nasally from the point of fixation a distance of five degrees, temporally as far as twelve degrees (and with Mariotte's blind spot added, a distance of from twenty to twenty-two degrees above), and below the point of fixation from eight to ten degrees. These statements hold good in the majority of cases where practical perimetry is resorted to. The scotomatous lesion is either complete, it being impossible not merely to tell the form of objects, but also to discern the colors, white, blue, red, and green, the defect in this case being called "scotoma absolutum," or it merely shows lack of color-perception for red and green, when it is named "scotoma relativum."

If the field of vision is disturbed in the manner described, it follows that the visual acuity will not only be disturbed centrally, but also will be more impaired temporally than nasally if both nerves are involved. Therefore each eye will be disturbed by itself in such a way that in looking at a row of horizontal objects, for instance at print, not merely the type directly in front of the eye will appear indistinct, but also the letters to each side, those on the temporal side being more blurred than those in the nasal half. Small colored objects, especially if green or red, or even larger ones seen at a distance (the images on the macula lutea being small when this is the case), lose their color and assume a grayish shade. In some cases only red and green tints are imperfectly distinguished, so that the patient's own hands or the faces of his friends or acquaintances seem to have a pallid, corpse-like tint, or, again, the colored tips of matches and samples of colored cloth have a gray and a pale appearance. It is claimed that light-perception is also somewhat disturbed. This is especially evident if Chibret's photometer be employed.

THE TWO CHIEF FORMS OF CHRONIC RETROBULBAR NEURITIS.

Retrobulbar neuritis is either chronic or acute. The chronic form may be subdivided into two chief clinical groups: intoxication-amblyopia, or toxic amblyopia, and retrobulbar neuritis proper.

Toxic amblyopia, due to the use and abuse of tobacco, alcohol, and stramonium (asthma cigarettes), etc., manifests the following characteristics: Appearing between the ages of thirty and fifty years, seldom later, in men who sleep little and smoke much, or at least use tobacco in some form, and at the same time indulge in alcoholic excesses and show evidences of disturbances affecting the digestive functions (although this is not always the case), there develops gradual dimness of vision in both eyes. The patient sees better in twilight, and seeks subdued light for his work. After a few weeks or months, inability to do close work manifests itself, this being the

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period at which the patient sometimes states that the visual disturbance suddenly took place. As a rule, the sight is impaired to the same extent in both eyes; it seldom sinks below $\frac{6}{60}$ (generally reaching $\frac{6}{24}$); a relative scotoma for red and green appears, showing an oval inclination. It is only in rare cases, especially in incurable relapses, that there exists around the point of fixation, for a distance of three degrees, a small absolute scotoma for all colors. The peripheric perception for white as well as for the rest of the colors is normal, so that around the scotoma a well-preserved broad band of colors can be determined. In exceptionally severe cases it may happen that in certain sections, in particular in the upper temporal quadrant, this band vanishes as if it had been broken through by the relative scotoma. With abstinence from tobacco and alcohol, color-perception returns in about fourteen days, and the scotoma again assumes its oval reclining form.

Pupillary disturbances are rare, the pupils being rather dilated than otherwise; it is unusual to find reflex rigidity of the pupils, and it is also rare to meet with any disturbance of the muscles of the eyes. The ophthalmoscopic appearance is negative, or there may be slight hyperæmia and discoloration of the papilla (more often nasally). Later there develops a dull pallor of the temporal wedge-shaped segment of the papilla, giving this portion of the nerve-head the tint of unglazed porcelain. The prognosis is always favorable, except, perhaps, in those disputed cases in which it is claimed that atrophy of the papilla has resulted.

In the case of a manufacturer of liquors who drank and smoked immoderately, and then began to confound the colors of the red and green samples of liquor in the flasks on the floor, the writer discovered, in addition to the symptoms enumerated, haziness of both lenses, the crystalline lens appearing as if a glass lens had split in a radiating direction and had become discolored in the cracked portions. A physician advised him to return for a cataract operation after a month. Under abstinence the transparency of the lenses was completely restored. The patient was kept under observation for a period of five consecutive years.

Retrobulbar neuritis proper often appears before the thirtieth year, more frequently among women, usually in one eye, and suddenly, at times accompanied by pain in the eye, particularly on moving the globe and on pressure. Visual acuity may, and generally does, sink below $\frac{6}{60}$. The ophthalmoscopic picture in the beginning may be either negative or show marked hyperæmia. Prominence of the papilla, reaching even one diopter, may be present, as well as discoloration of the margins of the disk, such as may be observed in intra-ocular neuritis at the commencement of choked disk. In this form grave lesions involving the pupils and the ocular muscles, as well as pronounced disturbances of the general system, are unusually frequent,—conditions the cause of which becomes apparent when the etiology of the disease is considered. This form of neuritis is most commonly an expression of multiple cerebro-spinal sclerosis, beriberi, diabetes,

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syphilis, lead poisoning, intoxication by sulphur-fumes in rubber-factories, and acute catarrhal affections.

Of special interest are the different neuritic inflammations of hereditary origin, which generally appear in the male descendants about the twentieth year. The causes are unknown ; in fact, in almost thirty per cent. no etiological factor can be discovered. In some rare cases a marked loss of blood has preceded the attack (gastro-hæmorrhagia). In two cases (Berger) a central scotoma was noticed in tabes in men who, it was asserted, neither smoked nor drank. In various other diseases, where the patient smokes or drinks much while in a reclining position, it may happen that the scotoma due to intoxication described above is accidental.

In an overwhelming number of cases, however, the central scotoma in these diseases differs entirely from the scotoma arising from poisoning by tobacco or alcohol. It is perfectly round, arranged around the point of fixation. It is often absolute, and extends as far as twenty degrees or more into the periphery (lead, sulphonal, diabetes, and sometimes multiple sclerosis). It may extend for the greater part below the point of fixation (syphilis, multiple sclerosis), or may assume a vertical, oval, or zigzag shape. The limitations for white and blue, as a rule, are normal; only in exceptionally severe cases is slight contraction present. In all there is subjective improvement in vision when illumination is reduced; perhaps because under these conditions the contrast between the central and eccentric images is not perceived. In mild cases the disease runs its course in a few weeks, and, where a relative scotoma existed, marked improvement in vision results. It may happen, however, that an extensive, absolute scotoma remains, at whose periphery blue may still be distinguished, while red in larger surfaces outside of the scotoma cannot be made out. In the great majority of cases blindness does not result.

In the course of weeks the ophthalmoscopic appearances change, and the reddened and discolored papilla becomes markedly pallid, or a brilliant white color may show itself in the temporal area of the disk.

Frequently, although to a less marked degree, the nasal half of the papilla becomes pale, so that the whole disk is of a lustrous white color, except the nasal portion, which is of a light rose-colored tint. The vessels, especially the arteries, are somewhat contracted; but often no contraction in the calibre of the vessels can be detected for years.

The writer has had under observation for five years two cases in men now thirty-five years of age, whose vision has become reduced to seeing fingers at a distance of three millimetres. There is an absolute central scotoma to the extent of thirty degrees. The papilla is a brilliant white, only its nasal edge being a rose-red. The temporal margin and the excavation are of almost a bluish-white lustre. On the other hand, the lamina cribrosa is distinctly visible here, and the vessels throughout have preserved their normal thickness. The pigmentation of the chorioid shows so few marks of any lesion that it would be easy to make a diagnosis of

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simple and not neuritic atrophy of the papilla. The general condition of the patients was and is at present normal. The cause of neuritis in these cases is unknown.

The prognosis for "restitutio ad integrum" is not a favorable one, as may be inferred from the preceding statements. In some rheumatic cases, in syphilitic lesions, and now and then in multiple sclerosis, very rarely in lead intoxication among type-setters, as long as no absolute scotoma amounting to ten degrees has appeared, we are justified in expecting the restoration of sight, or at least great improvement in vision. As a rule, however, when an absolute central scotoma once makes its appearance, even with the most approved treatment it is hardly possible to achieve any perceptible betterment in the visual acuity; this, in the writer's experience, also holds good for the absolute central scotoma which manifests itself in diabetes. Schmidt-Rimpler claims that a cure or improvement has been effected in cases of diabetes with a relative scotoma, if this was not due to tobacco or alcohol. As has been stated, the writer has not seen any improvement in diabetic patients whom he has sent for treatment to Carlsbad, not far distant from his own home at Prague.

As regards complete loss of vision, the prognosis is favorable, for it is only in exceptional cases that total atrophy of the optic nerve occurs. In cerebro-spinal sclerosis this scarcely reaches three per cent. (Uhthoff).

An unfavorable prognosis is to be expected if not only an extensive absolute central scotoma exists (along whose edges perception for green and red is disappearing, if it has not vanished), but also if the peripheric perception for white contracts, and if in spite of treatment neither the normal limits for white nor for red or green return. It is to be regarded as a most ominous symptom if perception for white not only disappears concentrically at the periphery but also is lost in sectors. In many places these peripheric gaps may combine with the central scotoma. In the rare cases of relative central scotoma which occur in older persons and which can hardly be demonstrated by means of very small colored quadrants (two millimetres), the patients merely complain of increasing difficulty in reading.

This disease, which has no demonstrable ophthalmoscopic features, progresses very slowly. The etiological factor may be attributed to an arterio-sclerosis of the ophthalmic artery in the optic canal. In this position the dilated atheromatous vessel presses on the optic nerve which rests upon the blood-vessels and produces a change in the curvature of the nerve, causing the nerve itself to assume a biscuit-shape (Otto) and its fibres to undergo partial atrophy.

In differential diagnosis it is necessary to take into account the rare but significant homonymous (paracentral) scotomata of central origin. In cases presenting such field defects the complaints likewise begin with difficulty in reading or counting; but, in distinction from the neuritic processes just considered, central visual acuity remains normal.

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Occasionally chorio-retinitis might prove misleading, especially at the start, when the changes are not clearly demonstrable by means of the ophthalmoscope. In these cases one usually sees punctate exudations in the upright image on the macula, the scotoma being a positive one, and perception of light being disturbed, which is not the case in true neuritis.

ACUTE RETROBULBAR NEURITIS.

This disease is distinguished from the preceding chronic form by its sudden and often fulminating inception. It is generally accompanied by severe pain in the eye, back of the eye, over the forehead, and in the head, as well as a rapid and marked disturbance in vision, which, commencing with dimness of sight, ends in total blindness in the course of one or several days, sometimes even in a few hours. Generally both eyes are affected. In less violent cases only one eye is involved, usually the left one. It is comparatively often found that the patients are at the same time suffering from encephalitis or myelitis, or from the sensory and motor manifestations of these lesions. Paræsthesia and paraplegia of the lower extremities are the symptoms most commonly met with (beriberi).

On account of the suffering which results, the patients refrain from moving the muscles of the eye, either the upper lid showing marked retraction or the lids being tightly closed. If the sufferer is asked to move the globe, lateral movements are executed imperfectly and with dread. Rolling of the eyes is more readily accomplished.

The slightest pressure on the globe elicits pronounced pain and reaction. If the amblyopia or the amaurosis is considerable, the pupils show great dilatation, and complete or partial failure to light-reaction is present. Ophthalmoscopic examination, and especially perimetric tests, are rendered very difficult. The ophthalmoscope shows the papilla or the fundus oculi to be in a normal state, or there may be only slight discoloration of the papilla, and perhaps somewhat distended vessels. In other cases the picture presented is that of intra-ocular neuritis and even of choked disk.

On account of its short duration, the choked disk in this instance exhibits only slight discoloration. The choked disk in retrobulbar neuritis also discloses no hemorrhages, or only insignificant streak-like extravasations of blood along the upper and lower margins of the swollen papilla. Under these circumstances the cloudy discoloration may extend into the retina. In particular a veil-like haziness may be detected here and there in the macula lutea, and in the surrounding locality rod-shaped, lighter-colored formations may be noticed.

Vision is generally reduced to from one-third to one-fourth, but it may be lowered to the mere ability to see to count fingers or may fall to complete blindness. Very often it can be proved that the external limits of the visual field are normal; only color-perception is wanting, this being explained by the scotoma, which is originally central, and at a later stage undergoes extension.

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In other cases it is claimed that concentric contraction of the visual field occurs. These statements, however, on account of the difficulties in conducting the examination of acutely suffering patients, are often unreliable. In the further course of the disease the amaurosis soon ceases; in a few days fingers are again counted, and with this the existence of a central scotoma is once more established. The papilla, which showed neuritis and swelling and appeared blurred, clears up, and its edges can be discerned. Paleness of the papilla, however, or at least pallor of its temporal portion, begins to manifest itself. Subsequently, complete restoration of sight may take place, or there may be a return to one-fourth or one-third of the former visual acuity. As a result of this defect, even in the most favorable cases, the existence of a former central scotoma can be established at a later period; and while colors are recognized, they are described as having a less saturated appearance.

It very often happens, however, that the papilla becomes completely atrophied and pale, while the blood-vessels suffer contraction. As a rule, the accompanying connective-tissue bands are not broad. The lamina cribrosa is generally not exposed. In these cases the amaurosis is permanent. According to Graefe and Elschnig, in cases where amaurosis in the beginning develops rapidly, especially when this is accompanied by choked disk, it can be stated that blindness or great amblyopia will result.

The result of the pathological investigations in a case of retrobulbar neuritis (Elschnig) shows that we are dealing with a primary interstitial neuritis. In his case, throughout the whole cross-section and the entire length of the nerve extensive infiltration of fat-granule cells took place, and the vessels became dilated, the same thing holding true of the pial sheath, especially for the layers adjoining the nerve. As a secondary manifestation explaining the visual disturbance, compression (in the beginning), then degeneration, of the nerve-fibres took place. In place of the bundles of nerve-fibres an exudation of glia-tissue, with multiplication of round and spindle-shaped cells, is found. The highest degree of this inflammatory infiltration is reached in the optic canal. A similar microscopic picture was discovered by Elschnig in the internal capsule of the brain, where symptoms of brain-disease were present. In the atrophic papilla, in addition to dilatation of the smaller blood-vessels, we meet with an increase of the cell-nuclei of the connective tissue. In place of nerve-fibres the bundles are composed of infiltrated connective tissue of the supporting framework, with here and there foci of round cells. Through the whole extent of the papilla whose tissue has undergone fibrillar degeneration, it is hardly possible to make out the presence of nerve-fibres in any place, the few left being mostly in the nasal half. The atrophy of the layer of nerve-fibres and of the ganglion-layer also invades the macula lutea. The outer surface of the pial and the inner surface of the dural sheath show plates of proliferating endothelium; occasionally both sheaths are connected near the globe by bands of wide-meshed granulation-tissue.

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In explanation of the origin of the inflammation, Hock has recourse to catching cold. Elschnig attributes the condition to toxic agents which at the same time attack the brain and the spinal column. In the writer's opinion, the painfulness might be attributed to rheumatic disturbances around and in the joints consequent upon catching cold.

As to the fact that the papilla in the ophthalmoscopic picture is normal in one case and swollen in another, Elschnig contends that this depends upon the extent to which the inflammation has penetrated forward. He also says that in case the process reaches the papilla, collateral œdema arises. The writer's view is that the swelling of the optic nerve reaches the threshold of the entrance of the vessels into the nerve, and in this way causes choked disk.

Albrecht von Graefe classes those cases in which the ophthalmoscopic picture demonstrates ischæmia of the papilla under the group of retrobulbar neuritis. Elschnig, on the other hand, asserts that these cases ought to be excluded, since amblyopia merely develops here without pain, and is slower in its course than in retrobulbar neuritis. A hemorrhage issuing from the central artery, or compression, might account for the ischæmia.

The subacute forms of interstitial neuritis merit particular attention. They likewise are accompanied by evidences of pain in and back of the eye as well as on pressure and on movement of the globe. They also lead to amblyopia or amaurosis of short duration, while the presence of an absolute central scotoma can generally be demonstrated.

At times, as simultaneous or as somewhat later manifestations, myelitic symptoms in the lower limbs may develop.

SUBDIVISION III.

PERINEURITIS.

In patients suffering from tubercular meningitis (Michel, Elschnig), or syphilitic meningitis (Uhthoff), or from suppurating meningitis, the inflammation advances into the nerve-sheaths. In such cases granulation-tissue will be found in the subvaginal space, the new formation penetrating into the nerve-substance from the pial sheath along the septa, but becoming more reduced the nearer it approaches the axis of the nerve, in this way differing from interstitial neuritis. In the small-celled infiltration of the pial sheath of the optic canal small hemorrhages and foci of small-celled infiltrates as well as microscopic tubercles have been found. The greater part of the subvaginal space is filled with small-celled infiltration and layers of proliferating endothelium derived from the three membranes. In suppurating meningitis the cavity is filled with leucocytes and fibrinous exudates. The subvaginal space may be obliterated in certain places, as, for example, in its anterior, ampulla-like portion.

In syphilitic meningitis Uhthoff found endarteritis of the ophthalmic artery. In suppurating meningitis, diplococci of Fraenkel have been seen

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by Weichselbaum. Elschnig has proved that the small-celled infiltration penetrated the dural sheath as far as the surrounding orbital tissue, while the process extended forward into the superficial layers of the sclera and as far as the perichorioidal space (sepsis?). In spite of this, the patient had no ocular pain or suffering on moving the eyes.

The ophthalmoscopic examination either gives negative results, with trifling discoloration of the papilla, or in some cases choked disk may appear (Uhthoff), this variation undoubtedly being produced by the same causes that have been considered in the section on interstitial neuritis and choked disk.

Visual acuity either remains normal for a long time or is variously affected, this depending mainly on the process which the granulation-tissue undergoes, as a result of which compression or atrophy of the nerve-fibres takes place. This generally occurs at the surface of the optic nerve,—a pathological fact which can be accounted for in view of the microscopic appearances. It is especially important to keep in mind that, in consequence of meningeal disturbances (for instance, ventricular dropsy, dropsical dilatation of the recess above the chiasm, etc.), a pressure may be exerted on the optic tract (the optic chiasm, and the optic nerve), producing at first compression and then atrophy of the nerve-fibres. Another cause of compression may be found in an engorgement of blood-vessels occurring in front of the dilated ventricles. Most commonly, however, in syphilitic gummatous meningitis the proliferating granulation-tissue, again falling prey to a process of degeneration, implicates the intra-cranial portion of the optic nerve and calls forth different conditions (varying like ebb-tide and flood-tide), which lead to various disturbances of visual acuity and of the field of vision (hemianopia, etc.), and at a later period to partial or total descending atrophy of the optic nerve.

The diagnosis is strengthened by the frequently occurring difference in the pupils, the disturbance of the ocular muscles, and the condition of the body in general.

In the treatment of retrobulbar neuritis and perineuritis, attention must first be directed to the disease which causes the ocular lesions (syphilis, tuberculosis, diabetes, lead intoxication, etc.).

In acute rheumatic forms, large doses of salicylate of sodium may be used (from four to six doses of one-half gramme each). If sweating is to be produced, this remedy may be combined with phenacetin (Bayer), each dose being placed in capsules, two capsules to be taken at night, within an hour of each other. In the interval hot tea and Priessnitz's compresses can be applied to the head. After this treatment the intense ocular pain and headaches often subside. In violent cases (even in the non-syphilitic ones) inunctions of gray ointment should be used. In desperate cases the salicylates can be combined with the gray ointment. The latter is contra-indicated, however, if distinct pallor of the papilla has manifested itself, in which case iodide of potassium should be employed alone. As a remedy

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against eye-pain and headache, sedative salves can be rubbed over the forehead and temples, and compresses saturated with Burow's solution may be resorted to. A dark room is also recommended. It is necessary to pay attention to the bowels and to the kidneys, particularly in myelitic complications.

PART III.

ATROPHY OF THE PAPILLA OF THE OPTIC NERVE.

NEURITIC ATROPHY.

Neuritic atrophy of the papilla, or papillitic atrophy, is that form of atrophy of the optic nerve which follows inflammation of the connective tissue. The increase and new formation of connective tissue cause the light to be reflected, and on this account the papilla appears like a shining and, in more recent cases, a dull white disk. At a later stage the nerve-head undergoes a seeming reduction in size, as though it were shrinking. The lamina cribrosa is invisible, or at most can be made out in those cases only in which the neuritic process has been situated chiefly behind the globe. As a rule, the temporal portion of the disk is much whiter and shows a slight excavation. The blood-vessels are contracted and are frequently bordered by broad white streaks. A characteristic appearance is presented by the area surrounding the papilla, in case this section has been previously swollen, so that the inflammatory activity has extended into the retina (choked disk). Under these circumstances the pigment will be found piled up abnormally around the margins of the papilla. A sectional view shows that the nerve has become remarkably thin, its sheaths are tremulous, and on microscopic examination the septa will be found dilated. The nerve-fibres within the septa have become disintegrated and effaced, leaving a fibrillar mass with numerous myelin globules and multiplication of cell-nuclei in their place.

Greeff has demonstrated (according to the method of Golgi and Ramón y Cajal) the existence of numerous multipolar glia-cells, which differed in structure from those of the normal nerve. Through the whole cross-section nerve-fibres are often discovered still in a state of preservation. This serves to explain why at times, although the ophthalmoscopic examination apparently discloses total atrophy, a striking degree of visual acuity may exist.

Concentric contraction of the field of vision for all colors usually occurs, or the periphery remains normal and a broad absolute central scotoma develops. As an ominous symptom, sector-like defects arise, extending from the periphery to the centre, the gaps being first noticed when searched for with green and red colors. As a rule, we may consider the prognosis unfavorable if the perception for green diminishes or disappears rapidly while at the same time the visual field for the rest of the colors begins to show contraction.

As regards loss of sight, see Atrophy after Retrobulbar Neuritis.

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Atrophy after papillitis, or neuritic atrophy, has also been called "ascending." Pathological researches, however, show that the neuritis involves the greater portion of the nerve behind the globe, and even invades the optic canal and the intra-cranial division of the nerve, so that we are plainly not dealing with a purely "ascending" process. Strictly speaking, the term "ascending" applies only to atrophy of the nerve-fibres if the process that leads to blindness takes place within the globe or the orbit, no matter what the cause may be, and after enucleation as well. In distinction from this, under "descending" atrophy must be included all the degenerative changes that result from the destruction of the intra-cranial or the intra-canalicular portion of the optic nerve. The following morbid processes may be considered among the exciting causes: pressure of brain-tumors or of new growths springing from adjacent bones, extensive accumulation of fluid in the ventricles, aneurisms, hemorrhages into the sheaths of the optic nerve, fractures of the base of the skull continued into the optic canal, granulating syphilitic tissue at the base of the brain or in the optic canal, and division of the optic nerve itself.

In reference to the diagnosis and prognosis in these cases it is particularly important to remember that if the injury (for instance, a hemorrhage or a fracture) is situated far behind the globe, notwithstanding the marked disturbance of vision, the fundus oculi may present a normal appearance ophthalmoscopically, and may remain so for several weeks, so that the absence of demonstrable changes may lead to the diagnosis of simulation. It is to be noted in these cases that the pupil is usually dilated and that the iris fails to react to light.

Fractures involving the optic canal have also been recorded after blows upon the face with a blunt instrument, and in one case after a fall on the ischial tuberosity without any serious visible injury. In a case under the writer's observation, in which, as a result of a blow (with a wedge) on the left side of the forehead, bleeding from the nose continued for several days, visual acuity became reduced to one-sixth, and was accompanied by manifestations of traumatic neurosis, notably mental depression, headache, etc. After eight weeks, the ophthalmoscopic appearance of the fundus was still nearly normal. The field of vision, however, began steadily to contract, and with this atrophy of the papilla developed, the disk, although rose-red in color, becoming smaller in size and less firm in structure.

After shot-wounds of the orbital cavity above the eye, complete blindness follows immediately, even though the optic nerve remains uninjured and the bullet does not lodge in the orbit. It is not until several weeks have elapsed that atrophic discoloration commences. At first the papilla may be normal in appearance.

It sometimes happens, after compression of the optic nerve in the cranial cavity, that ophthalmoscopic examination discloses total atrophy of the papilla, while at the same time vision is surprisingly good. This is especially true in the so-called "boat-shaped" and "steeple-shaped" malforma-

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tions of the skull, where, for instance, very acute sight may exist although the papilla is pale and atrophic. In these cases nystagmus is a frequent attendant symptom, and convulsions during infancy are common. This partial atrophy may often be explained by hyperostoses of the bones of the skull and narrowing of the bony optic canals. Many of these atrophic conditions, however, are the sequelæ of neuritic processes which have run their course under the guise of intra-ocular neuritis or choked disk.

The writer can never forget a certain case (in a man thirty-five years old) in which both papillæ, but particularly the left, were totally white. The vision in the right eye was normal, and in the left eye it was reduced to the ability to see to count fingers. Under the use of iodide of potassium the patient began to see sufficiently to be able to read with the aid of the left eye. Two years later death ensued as the result of an epileptiform seizure (cerebral syphilis?).

The degeneration following compression and laceration of the nerve approaches the so-called progressive gray atrophy of the optic nerve.

The papilla in progressive gray-white atrophy, in contradistinction to its appearance in neuritic atrophy, has sharply defined outlines; the surrounding pigment is regularly arranged; the area of the disk is not decreased; the blood-vessels remain normal and unaltered in calibre for a long time, and the lamina cribrosa can be made out by means of grayish marks and dots in the bluish or greenish gray-white surface of the nerve-head.

Examination shows that the nerve itself does not become as attenuated and thin as in the neuritic inflammation. On section it merely appears grayish in color and seems somewhat translucent. Microscopically, the markings of the septa are well preserved, and no trace of interstitial inflammation can be detected. On the other hand, it is plain that we are dealing with a primary atrophy of the nerve-elements, these perhaps first undergoing disintegration. The nerve-fibres are transformed into extremely fine fibrillæ that stain with carmine. It is not until very late that the septa undergo thickening and sclerosis. Similar changes take place in the walls of the smaller blood-vessels.

The fibres that first give way to the degenerative process are those that are situated on the surface of the optic nerve. This degeneration may appear in several different places, a fact which accounts for the different peripheric alterations in the visual field. The most common cause of this degeneration is tabes dorsalis. According to Gowers, lesions of the optic nerve are found in thirteen per cent. of all cases of this disease; according to Berger, in thirty-three per cent.; and according to Dillmann, in forty-two per cent. It would be advisable to have statistics collected in the general and ophthalmic clinics of cities far apart from one another, since patients of this class often consult several physicians and in this way a double record is obtained. At all events, the records of the ophthalmologist will always show a higher, and those of the general physician a lower, percentage of "optic atrophy."

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According to Gowers, gray degeneration of the optic nerve occurs in thirty-five per cent. of tabetic patients, according as these at the same time exhibit psychical disturbances. The atrophy most frequently arises in the preataxic period; it may at times, however, begin when ataxia has developed; and it may even take place during the paralytic stage. It is rather commonly combined in progressive paralysis with sclerosis of the posterior columns of the spinal cord. Regarding the numerous aids to the diagnosis of tabes furnished by ocular and neurological symptoms, reference is made to the articles (vol. iv. of the System) on the interdependence of diseases of the eye and diseases of the general system. Here also will be found enumerated various other etiological factors assisting in the production of atrophy of the optic nerve, such as general, internal, and gynæcological lesions, and especially infectious diseases. Many of these atrophic processes could, however, hardly be classed with "gray degeneration of the optic nerve."

A chief symptom in differential diagnosis is that in gray tabetic degeneration a central scotoma is never found. Berger alone records in more than one hundred cases a central scotoma in tabes where the patients were not smokers or drinkers. In this form of atrophy occasional cases are met with in which the ophthalmoscopic condition of total atrophic paleness of the papilla is not accompanied by a corresponding reduction in vision, but, on the contrary, visual acuity is preserved in a surprising degree. At most the patients complain that their sight is dim. Atrophy of the papilla is often seven years (Kohler), ten years (Charcot), or from fifteen to twenty years (Gowers) in advance of the development of other symptoms, so that many cases of atrophy which have been considered genuine for years prove to be either those exhibiting prodromic symptoms or those manifesting but an isolated symptom of tabes.

The atrophy affects both eyes, at times appearing first in one and later, even after a number of years (very rarely), in the fellow eye. Visual acuity decreases steadily and slowly (from two to three years). It is claimed that in some cases blindness has not appeared until after from ten to twenty years' time. Sometimes in the progress of the disease slight and unreliable improvement occurs or a temporary arrest takes place. Loss of sight within a few months is one of the rarest occurrences (Hirschberg).

As the central visual acuity decreases, the field of vision may undergo oval contraction. Color-perception, first for green, disappears, and sector-like defects arise. Occasionally an eccentric portion of the visual field, especially to the temporal side, remains unimpaired for some time. As a rule, however, the defect shows itself in the temporal quadrant.

As to prognosis, it is important to remark that those instances in which the atrophic pallor of the papilla seems to be surprisingly advanced while the visual acuity remains remarkably good belong to the cases in which the fixed condition continues for a long time or the progress is very slow. The same is true in cases in which the visual field for colors shows a contraction corresponding to the limitation which white undergoes. On the other

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hand, if the field for white remains comparatively normal while strikingly rapid contraction for colors takes place, or perception for green vanishes, or a sector-like defect for colors appears, we may infer that the progress of the atrophy will be rapid (Knies). Uhthoff describes a sector-shaped, non-progressive atrophy of the optic nerve as occurring in a case of tabes.

Therapeutically, hydrotherapy still seems to offer the best results; internally, silver nitrate is of use. Electricity (Knies) and gray ointment (Albrecht von Graefe, Knies) are contra-indicated. Blindness more rapidly results under influences that weaken mind and body: therefore strengthening and nourishment of the general system are necessary. It is worth mentioning that on ophthalmoscopic examination we sometimes encounter mixed forms, a combination of post-neuritis and gray degenerative atrophy (for instance (Vossius), where the lamina cribrosa in particular is not distinctly visible). On the other hand, we may meet with a lamina plainly visible after a number of years, as well as normal vessels, in cases that follow a neuritic process which ran its course back of the globe and did not reach the papilla. The absence of an absolute central scotoma in simple progressive atrophy will generally serve as a guide against error.

It is often impossible to establish a positive differential diagnosis between atrophy of the papilla in pseudo-tabes of syphilis and tabes. Intense headache, apoplectiform seizures, polyuria, polydipsia, and frequent miscarriages point to atrophy due to syphilis, during which bitemporal hemianopsia and permanent disturbances of the ocular muscles are common occurrences. In recent times any history of miscarriages must be considered cautiously, since miscarriages artificially produced are increasing. In favor of tabes may be cited transitory disturbances of the muscles of the eye, particularly the rapidly disappearing ptosis, as well as sudden loss of accommodation without mydriasis and a marked tabetic flow of tears.

Occupying a middle position between post-neuritic and tabetic atrophy is atrophy accompanying multiple cerebro-spinal sclerosis. It will be remembered, however, that complete atrophy appears in this disease in only three per cent. of cases (Uhthoff). In the remaining ones the papilla either is without any especial changes (forty-eight per cent.) or shows only partial pallor (nineteen per cent.). It may be merely somewhat paler in the temporal portion, as in toxic amblyopia, or only whiter (eighteen per cent.). In six per cent. the process runs its course under the form of an intra-ocular neuritis.

The pathological change in the sclerotic foci of the optic nerve partly resembles that met with in neuritis and that found in simple degeneration. The explanations for this mixed process vary. "The process begins in the more delicate connective-tissue septa, and only at a later stage affects the coarser ones. Atrophy of the nerve-substance is secondary. Disintegration and disappearance of the medullary sheaths are rapid and complete, while the axis-cylinders often remain permanently preserved."

An important point in differential diagnosis is that in the cases unac-

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companied in the beginning by any perceptible ophthalmoscopic changes, a central relative or absolute scotoma exists. For this reason the disease has already been considered under retrobulbar neuritis.

The experienced diagnostician will be further protected from error by the peculiar, spotted, cold-looking pallor of the papilla in advanced cases. Other attendant symptoms are increase in the patellar tendon reflexes, tremor of intention, tremor of the muscles of the face, and nystagmus.

Under "atrophy of the optic nerve" it is usual to group affections of the nerve arising after great loss of blood, especially after gastrorrhagia or metrorrhagia. The same condition may appear after puncture of large ascitic masses of fluid, and after vomiting. In very few cases of the sanguineous variety blindness is present as soon as the patient awakes from the syncope following the hemorrhage. Far more frequently complaints regarding dimness of vision or marked visual disturbances fail to appear until three or four days have passed; in comparatively rare cases as late as twelve or fourteen days. Both eyes are affected, the irides do not react to light, and the fields of vision show peripheral scotomata. Ophthalmoscopic examination discloses at the very start ischæmia of the artery. The veins are overfilled, the papilla is discolored, appears moist, and often projects forward. It is also common to see the outlines of the disk blurred and to find small hemorrhages in the retinal area adjacent to the margins of the papilla. According to Friese, in an average of a hundred cases of post-hemorrhagic blindness, fifty ended in complete atrophy of the optic nerve, twenty in recovery, and thirty in improvement. In the latter cases only a small peripheral portion of the visual field remained.

Ziegler has examined the optic nerve twenty-five days after the inception of the condition, and discovered a fatty degeneration of the optic nerve-fibres in the retina and in the optic nerve.

PART IV.

CONGENITAL ANOMALIES AND TUMORS OF THE OPTIC NERVE.

For "Congenital Anomalies of the Optic Nerve" the article on "Congenital Malformations and Abnormalities of the Human Eye," by William Lang and E. Treacher Collins, in vol. ii. of this System, can be consulted.

Primary tumors (Figs. 6, 7) of the optic nerve are extraordinarily rare. As a rule, they belong to the benign myxomata and occur in childhood and youth (seventy-five per cent.). They develop painlessly and very slowly within the unaffected dural sheath, producing hypertrophy and dilatation of the pial sheath and the subvaginal space. Assuming a bulbous or a band-like form, they penetrate into the optic nerve, producing atrophy of

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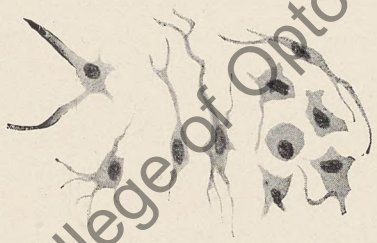
FIG. 6.

Primary neoplasm of the optic nerve; benign myxoma; cross-section of a myxomatous optic nerve (low power); hardening by Müller's fluid. Stained with hæmatoxylin.

FIG. 7.



Myxoma of optic nerve (high power).

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the nerve-fibres. These neoplasms, somewhat resembling a post-horn in shape, attain a size varying from the diameter of a few millimetres to that of an egg. They are hard or have a soft cartilaginous or a spongy consistency, and on section compartments are frequently found filled with collections of mucus.

The most common site of the new growths is at the anatomical seat of the entrance of the blood-vessels into the nerve (from six to seventeen millimetres behind the sclera). Rather often they extend through the optic canal (which becomes distended) as far back as the optic chiasm. Not infrequently the optic nerve of the other eye is affected. The tumor very rarely reaches the posterior orbital wall, this extension in growth being first evidenced by flattening (acquired hypermetropia) and then by destruction of the globe.

The most common symptom is exophthalmos, which takes place forward and a little to the outer lower side. Blindness or marked visual disturbance arises early but slowly, and sometimes even before the appearance of exophthalmos (rare). Usually it sets in with the beginning of protrusion. Ophthalmoscopically all stages of neuro-retinitis and choked disk are met with, the papilla in the latter cases at times showing an elevation as high as two and a half millimetres (Hessdoerfer). Tillane mentions the occurrence of great pain on illumination of the unaffected eye.

Microscopically it will be found that the myxomata (Fig. 7) consist mainly of round cells that are unequal in size, and of stellate cells with long projections wound spirally.

After extirpation, and even with preservation of the globe (which later becomes atrophied), generally no return of the new growth occurs, even when a part of the neoplasm has been left in the optic canal. The time of observation after the operation, however, has not exceeded a few years, —a fact which must not be overlooked when we remember the long term of years which characterizes the development of the growth.

In differential diagnosis, the rare, encapsulated, cavernous tumors which develop painlessly for years along the course of the optic nerve, but which do not lead to blindness until very late in their progress, must be considered.

Much more dangerous are the neoplasms of the external sheath of the optic nerve, which have started in the sheath or perhaps have passed over into it from the orbital tissue. These grow rapidly, endanger sight at rather a late stage, and after incomplete removal are followed by a rapid and more malignant reappearance. They are composed of epithelioid cylindrical or cubo-cylindrical cells, arranged in tubes, cylinders, and interbranching twigs in such a way that the cells occupy a vertical direction to the tube, whose centre is penetrated usually by new-formed thin-walled vessels. Epithelioid and spindle-shaped cells are also met with. Very often hyaline degeneration may appear in numerous places. In some cases there is even calcification.

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These tumors belong to the group of endothelioma-angio-sarcoma, and correspond to the kind of cells which predominate. In literature they have been described under the most varied names.

In Taylor's case an endothelial sarcoma penetrated into the interior of the eye. Primary glioma of the orbital optic nerve has also been described. Outside of the secondary new growths, which extend into the optic nerve from the inner part of the globe or from the orbit, mention must be made of the very rare form of tuberculosis of the optic nerve, in which the nerve was found swollen cylindrically, and where the existence of clusters of tubercles in the retina had been demonstrated (Sattler[1] and Cirincione[2]). Hirschberg[3] and Scheidemann[4] have described cases of gummatous new formation behind the eyeball and in the papilla with recovery or marked improvement after treatment by inunctions.

Metastatic carcinomata (primarily ovarian) of both optic nerves behind the sclera have been mentioned by Krohn.[5] A case of metastatic sarcoma of the papilla (originating in the nævus of the sternum) has been published by Schiess and Roth.[6]

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[1] Archiv für Ophthalmologie, xxiv. 3.
[2] Annali di Ottalmologia, xix. 1.
[3] Eulenburg Realencyclopaedie, Ophthalmoscopie.
[4] Archiv für Ophthalmologie, xli. 1.
[5] Klinische Monatsblätter für Augenheilkunde, x. 103.
[6] Archiv für Ophthalmologie, xxv. 2.

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GLAUCOMA: PATHOGENESIS, SYMPTOMS, COURSE, AND TREATMENT.

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Glaucoma is a complex morbid process depending essentially on an excess of pressure in the chambers of the eye. The initial causes of this excess are very various, but they culminate in all cases in an obstruction of the escape of the intra-ocular fluid; hence the increased tension of the eye which is the leading symptom of glaucoma. The other symptoms are due to the excess of pressure, and vary much in different forms of the disease. In the acute they are urgent and severe, including intense congestion, violent pain, and rapid loss of sight; in the chronic they are at first slight and insidious, and may for a long time be unnoticed, even by the patient. In all forms, unless relieved by timely treatment, they end in total and permanent loss of sight.

The name glaucoma, as originally used, referred to a discoloration of the pupil sometimes seen in the later stages. The overfulness of the eye was little noticed until the year 1830, when William Mackenzie called attention to it, and made the first attempt to relieve it by puncturing the tunics. Twenty-five years later Von Graefe, studying the subject afresh with the aid of the ophthalmoscope, convinced himself that the increased pressure in the eye was no mere complication, but was the cause of all the leading symptoms of the disorder, and was thus led to his beneficent discovery of the cure by iridectomy.[1] The name, therefore, now no longer implies anything as to the appearance or condition of the pupil; it implies that the eye is suffering from undue pressure in the chambers, and it is properly applied to every eye thus affected, whether it be suffering at the same time from other manifest disease or not.

Glaucoma is called *primary* when it does not appear to have been caused by previous disease in the eye; *secondary* when it does appear to have been so caused. Some cases occupy a doubtful position, but, as a rule, the dis-

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[1] An admirable outline of the history of the subject may be found in Snellen's Historical Essay on the Development of our Present Knowledge of Glaucoma, Ophthalmic Review, 1891, p. 33.

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tinction is easily made, and is important. The primary forms exhibit the typical symptoms more clearly than the secondary, for in the latter the associated disease often masks or modifies the special pressure-changes; but it is important to note that, apart from their initial causes, primary and secondary glaucoma are to a large extent the same morbid process. All the symptoms which belong to primary glaucoma are to be found in one or other form of secondary glaucoma also.

One of the results of persistent pressure in the eye—excavation of the optic disk—is occasionally seen in eyes of normal tension, and on this account some writers describe a form of glaucoma which is not dependent on increase of pressure in the eye. But this leads to confusion of ideas and of terms. The excess of pressure which suffices to cup the disk is sometimes very slight and often intermittent; it happens, therefore, that eyes suffering from true glaucoma present at certain times a strictly normal tension, or an excess which is hardly discoverable. On the other hand, there is reason to believe that an excavation closely resembling the glaucoma cup may arise through simple atrophy of the nerve in conjunction with a physiological excavation. Such a condition, when definitely diagnosed, should be excluded from the glaucoma group. (See p. 661.)

PATHOGENESIS OF GLAUCOMA.

The study of glaucoma demands a knowledge of the processes by which the supply of fluid to the chambers of the eye is maintained and regulated. The mystery which continued to surround the causes of the disease long after its dependence on increased pressure had been recognized was due chiefly to the want of this knowledge.

Secretion, Excretion, Composition, and Pressure of the Intra-Ocular Fluids.[1]—The secreting surface of the ciliary body supplies a fluid which nourishes the vitreous and the lens and replenishes the aqueous chamber.[2] The fluid passes for the most part forward through the pupil into the anterior chamber, and escapes thence by filtering through the ligamentum pectinatum into Schlemm's canal and the veins connected with it. A very much smaller quantity passes slowly backward through the vitreous, and probably finds an exit by the perivascular spaces in the optic nerve. Whether there is any continuous stream from the vitreous into the aqueous chamber is doubtful, but it is certain that the hyaloid membrane is readily permeable, and that in the healthy eye any excess of fluid in the vitreous chamber finds ready exit in this direction.

The fluids of the aqueous and vitreous chambers are nearly identical in composition: water about ninety-nine per cent., salts and extractives about

[1] For a fuller account of the physiology of the subject, with references to the more important investigations and papers, see author's Erasmus Wilson Lectures on the Pathology and Treatment of Glaucoma, London, Churchill, 1891, pp. 13–29.

[2] For the minute arrangement of the secreting cells, see a paper by Treacher Collins, Trans. Ophth. Soc. of United Kingdom, vol. ii. p. 55, 1891.

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one per cent., albumen in quantity too small for estimation.[1] The fluid which refills the anterior chamber after it has been emptied by puncture of the cornea contains albumen in considerable quantity, for under the sudden lowering of the intra-ocular pressure the capillaries of the ciliary body enlarge, the secreting epithelium fails in its selective action, and the albuminous constituents of the blood exude.[2] Similarly, when the ciliary body is inflamed its secretion often becomes highly albuminous. This change in the constitution of the intra-ocular fluid plays a part in many forms of glaucoma.

The intra-ocular pressure equals that of a column of mercury about twenty-five millimetres in height, and presents no discoverable difference in the aqueous and vitreous chambers respectively. In an optical instrument built, as the eye is, of soft material, such internal pressure is indispensable. It keeps the tunics in a state of moderate tension, and thereby maintains the form of the globe and gives precision to the action of its muscles. It is so regulated as not to embarrass the circulation of the blood or the nutrition of the tissues within the organ. When regulation fails and the pressure in the chambers rises above physiological limits, we have the complex disturbance of function and structure called glaucoma.

The intra-ocular pressure fluctuates with the pulse and with the movements of respiration; it rises slightly during contraction of the external eye muscles; it varies with the force of the blood-stream, and is under the influence of the fifth nerve. These variations have been established by experiment in the case of certain of the lower animals, and doubtless occur also in the human eye. They appear to be always transient, for so long as the escape of fluid from the chambers is unimpeded, a rise of pressure is soon compensated by an increased outflow. A persistent excess of pressure appears to be associated in all cases with an impediment to the outflow.

Retention of Intra-Ocular Fluid.—In 1876, three years after Leber had demonstrated the escape of the aqueous fluid at the angle of the anterior chamber,[3] Max Knies and Adolph Weber, independently of each other, showed that in eyes blinded by glaucoma this outlet is commonly obstructed.[4] A peripheral adhesion of the iris in such eyes had been incidentally noted by previous writers, but its significance was now recognized for the first time. Since that time multitudinous forms of glaucoma have been studied with regard to the same point, and in all forms, without exception, changes likely to obstruct the escape of fluid at the filtration-angle have been found. Moreover, in a considerable number of eyes blinded by glaucoma, the permeability of the filtration-angle has been tested by injection of fluid into the anterior chamber, and in every instance a great

[1] Michel and Wagner, Physiolog.-chem. Untersuchung. des Auges, Von Graefe's Archiv, xxxiii., ii., S. 155, 1886.

[2] R. Greeff, Interchange of Fluids in the Eye, etc., Report of Heidelberg Ophth. Soc. for 1893, p. 11; and Ophth. Review, 1894, p. 22.

[3] Von Graefe's Archiv, xix., ii., S. 87.

[4] Ibid., xxii., iii., S. 163, and vol. xxiii., i., S. 1.

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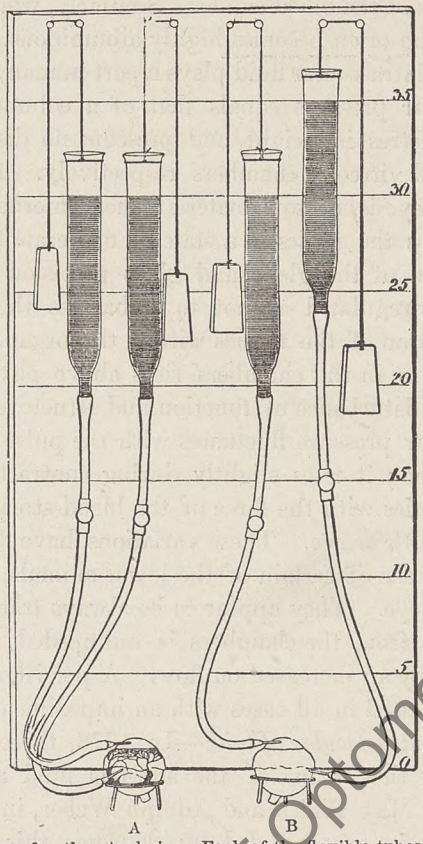
impairment of filtration has been found.[1] It is perhaps not surprising that the so-called retention theory of glaucoma is not yet accepted by all ophthalmic surgeons, for the most acute observer, studying the matter only from the clinical point of view, sees nothing of the normal filtration-

FIG. 1.



Injection apparatus, one-fourth actual size.—Each of the flexible tubes is provided with a pinch-tap, and ends in a hollow needle. The fluid is a one per cent. solution of aniline black. Air is carefully expelled from the tubes. The eyes of a sheep are removed immediately after death and injected while still warm, both at the same time. In each eye, one needle enters the aqueous chamber, the other the vitreous chamber. The aqueous tube is opened a few seconds before the vitreous tube, so as to insure a full anterior chamber at starting.

Eye A.—The two columns stand at the same level, thirty centimetres above the eye. The pressures in the aqueous and vitreous chambers are equal; the lens and iris are not displaced; the filtration-angle remains open; the fluid quickly injects the episcleral veins and flows from their cut ends.

Eye B.—The vitreous column stands at thirty-five centimetres, the aqueous at thirty centimetres above the eye. The vitreous pressure is a little higher than the aqueous pressure; the lens and iris advance; the anterior chamber is shallow, the filtration-angle is closed; there is no injection of the episcleral veins and no escape of the colored fluid at any part of the eye, even after several hours.

The condition of the filtration-angle in each case may be ascertained by freezing the eye while the injection is in progress and dividing it. (For measurements of amounts of fluid passing through the chamber, see Ophth. Rev., 1887, p. 193.)

process and little of the special conditions which disturb it in glaucoma; but for those who have investigated the subject thoroughly in the labora-

[1] Leber and Bentzen, Von Graefe's Archiv, xli., iii., S. 208.

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tory as well as in the consulting-room, the evidence is complete that retention is the essential factor in the morbid process.

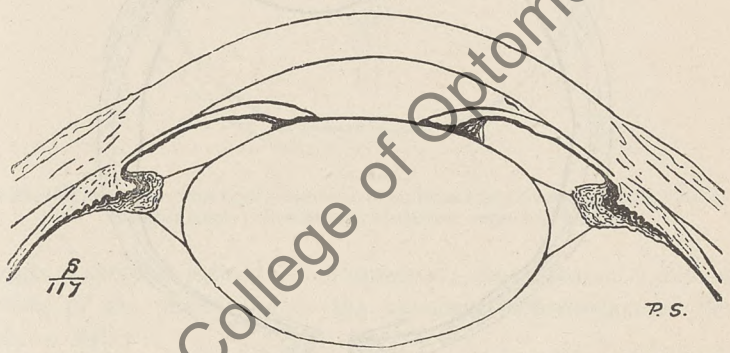
Fig. 1 exhibits an injection experiment which demonstrates in a striking manner, first, a free escape of fluid from the anterior chamber into the episcleral venous plexus, and, secondly, a total arrest of this escape when the angle of the chamber is closed. By varying the pressures it can be shown that a slight compression of the filtration-angle not amounting to closure retards without arresting the escape of fluid from the chamber. The manner in which the filtration-angle is closed in this experiment closely resembles what occurs in some of the chief forms of glaucoma.

Initial Changes.—The obstruction at the outlet, discoverable in eyes blinded by glaucoma, explains the excess of pressure in the chambers, but it does not reveal the starting-point of the disease. What causes the obstruction? The initial causes of glaucoma are very numerous, and it will be well to describe first the sequence of events in various forms of secondary glaucoma, for in these the causation is more easily discerned than in primary glaucoma. The following descriptions are all taken from actual cases; the illustrations are reproduced from camera drawings of actual specimens. The methods which were employed in the preparation, examination, and preservation of these specimens have been described elsewhere.[1]

CAUSES OF SECONDARY GLAUCOMA.

Annular Posterior Synechia.—After repeated iritis the pupil-margin remains widely adherent. The surgeon, foreseeing a glaucomatous attack should the adhesion become complete throughout the circle, proposes iridec-

FIG. 2.



From an eye blinded by secondary glaucoma following neglected iritis.—Exclusion of the pupil; accumulation of fluid in the posterior chamber; displacement of the iris; closure of the filtration-angle.

tomy, but the patient refuses, for the tension is normal and there is no pain. A little later the patient returns, the eye being now hard, painful, and injected. The exclusion of the pupil has become complete. The transit of fluid from the posterior to the anterior chamber is stopped; the

[1] Author's Lectures, Appendix, p. 182; and Ophth. Review, 1894, p. 218.

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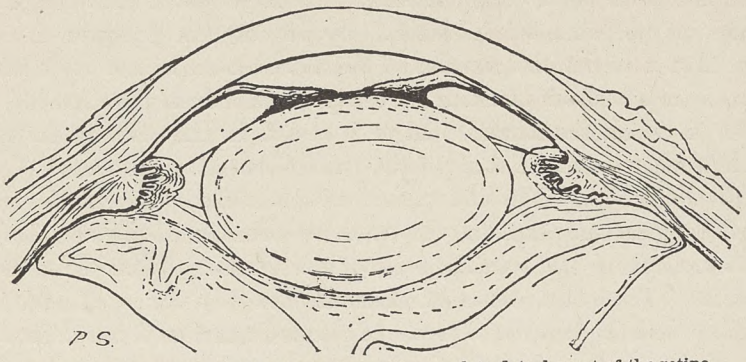
iris is pushed forward by the accumulation behind it; the filtration-angle is abolished. A small iridectomy is made at once, the communication between the chambers is re-established, the iris retires to its normal posi-

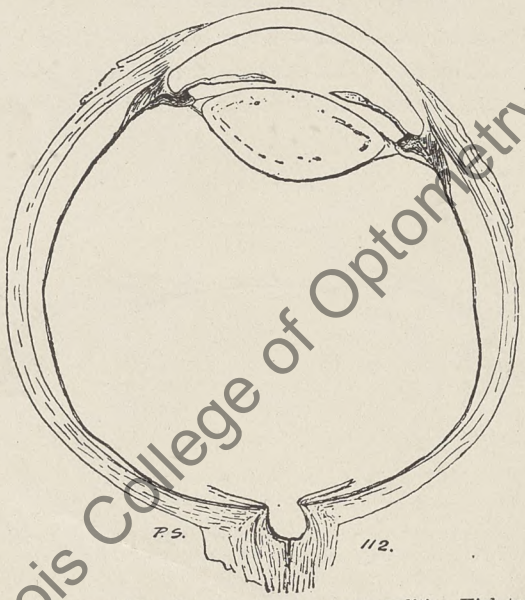
FIG. 3.



The same changes as in Fig. 2, together with complete detachment of the retina.

tion, and the glaucoma is gone; or the eye, being already blind, is excised, and we find the iris-base widely in contact with the cornea. (Figs. 2 and 3.) In eyes blinded by this form of glaucoma, complete detachment

FIG. 4.



From an eye blinded by secondary glaucoma due to serous cyclitis.—High tension due to serosity of the fluid and deposit in the aqueous chamber; filtration-angle distended; disk deeply cupped. (Specimen and notes from Mr. McHardy.)

of the retina usually supervenes sooner or later; and ultimately, in spite of the closure of the filtration-angle, the eye softens and shrinks,—a sign that the ciliary processes are atrophied and have ceased to secrete.

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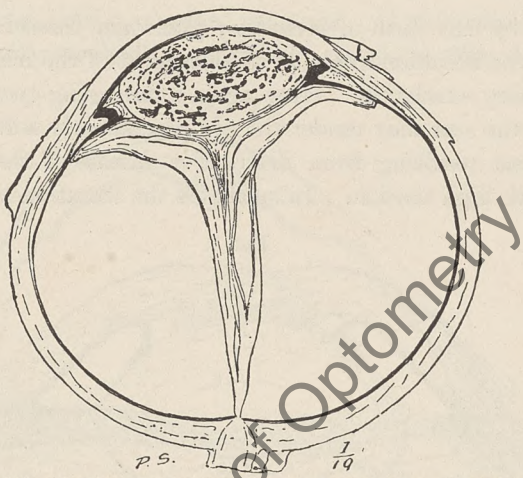
Total, like annular, posterior synechia may lead to secondary glaucoma. The fluid then accumulates behind the lens, the united iris and lens are driven forward together, and the anterior chamber is almost completely abolished.

Serous Cyclitis.—The inflamed ciliary body pours into the aqueous chamber a morbid albuminous fluid which escapes from the eye with greater difficulty than the normal secretion. It accumulates in the anterior chamber, displacing the lens and iris backward, depositing punctate opacities on Descemet's membrane, and clogging the filtration-angle. The tension of the eye is increased. Puncture of the cornea, by allowing the accumulated fluid to escape, relieves the glaucomatous condition at once, and, being repeated in case of need, together with measures directed against the cause of the cyclitis, permanently cures it. If the tension continue unrelieved, the eye becomes blind and presents an excavated disk like that found in other forms of glaucoma. (Fig. 4.) This form differs from most others in the fact that the anterior chamber is deep instead of shallow, the filtration-angle distended instead of compressed; the retention is due not to a narrowing of the outlet, but to the abnormal constitution of the fluid. (See also p. 645.)

FIG. 5.



From an eye lost by cyclitis after fever.—Secretion of aqueous and vitreous fluids suppressed; aqueous chamber empty; vitreous body atrophied; tension subnormal.

In higher degrees of cyclitis the eye becomes soft rather than hard, for the secretion process is impaired or suppressed. Then, together with shrinking of the vitreous and degeneration of the lens, we have complete abolition of the aqueous chamber. (Fig. 5.) In such an eye, although the filtration-angle is annihilated, high tension is impossible, unless it be due to rupture of a blood-vessel and outpouring of blood.

Perforating Wounds and Ulcers of the Cornea with Anterior Synechia.—The aqueous escapes through an aperture in the cornea; the iris applies

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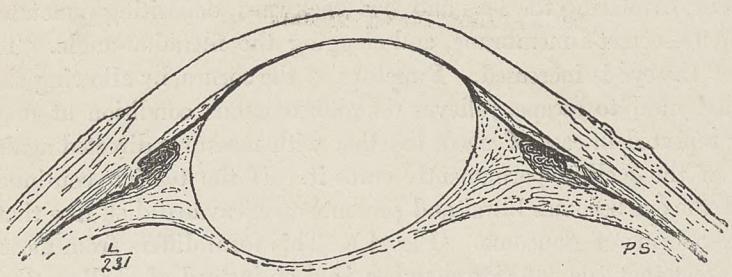
itself to the cornea and adheres to the wound. So long as the wound leaks, the eye remains soft; when this ceases it becomes hard, for the fluid has now no sufficient outlet at the filtration-angle. The lens is sometimes pushed forward so as to abolish the anterior chamber. (Figs. 6 and 7.) If it be involved in the injury, the ruptured capsule, the lens-substance, or even the vitreous may become adherent to the corneal cicatrix. (Fig. 8.) In

FIG. 6.

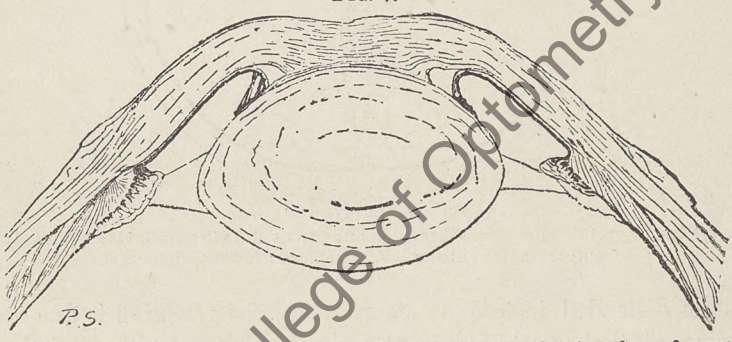


From an eye blinded by secondary glaucoma following jagged wound of cornea, not seen in section.—Permanent abolition of anterior chamber; access to filtration-angle cut off; inflammatory exudation in posterior chamber.

eyes blinded by this form of secondary glaucoma, dissection always shows abolition of the filtration-angle by displacement of the iris, often confirmed by inflammatory exudation. Here again a damaging cyclitis or a fistulous condition of the scar may render the eye permanently soft instead of hard.

Staphyloma resulting from destructive disease of the cornea is often associated with high tension. In such eyes the filtration-angle is abolished,

FIG. 7.



From an eye blinded by secondary glaucoma following central perforating ulcer of cornea.—Lens adherent to corneal cicatrix; pupil-margin adherent to lens; anterior chamber abolished; access to filtration-angle cut off; fluid imprisoned in posterior aqueous chamber.

for the iris is included in the pseudo-cornea and there is no anterior chamber. (Fig. 9.) Unless there is a leakage somewhere, high tension and progressive disorganization are the necessary results. In some cases there does appear to be a persistent or periodic leakage, due probably to a defect in the epithelial lining, which in the normal eye keeps the cornea water-tight. High tension is then either permanently absent or occurs only from time to time, subsiding when the leakage is re-established.

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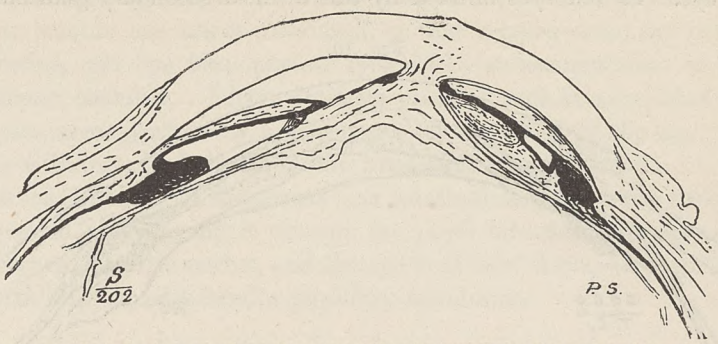
Cataract Operations.—The secondary glaucoma which occasionally follows cataract extraction is of special interest and importance. It may occur during the after-treatment, or after recovery is apparently complete, or even

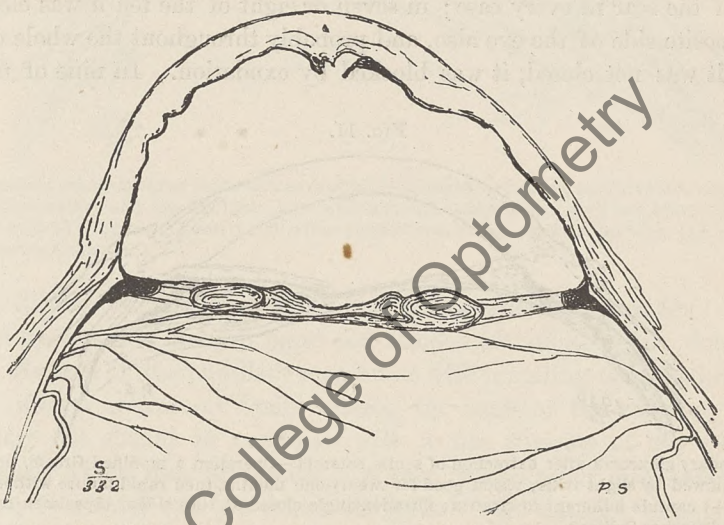
FIG. 8.



From an eye affected with secondary glaucoma following puncture of cornea and lens by scissors.—Synechia; adhesion of capsule and vitreous to corneal cicatrix; closure of filtration-angle; disk deeply cupped.

after a good result has been maintained for years. It is not absolutely avoidable by any particular method of operating. It occurs after simple extrac-

FIG. 9.



From the eye of a child blinded by secondary glaucoma following destructive ulceration of cornea.—Anterior chamber abolished as in Fig. 7; lens disorganized through adhesion with cornea and subsequent separation with rupture of capsule; corneal staphyloma and enlargement of globe (antero-posterior diameter, thirty-one millimetres; transverse, twenty-seven millimetres); disk deeply cupped.

tion without iridectomy; after extraction with iridectomy or preceded by a preliminary iridectomy; and after extraction in the capsule.[1] It appears to depend in all cases on some interference with the patency of the filtration-

[1] Natanson, Ueber Glaucom in aphakischen Augen, Mattieson, Dorpat, 1889.

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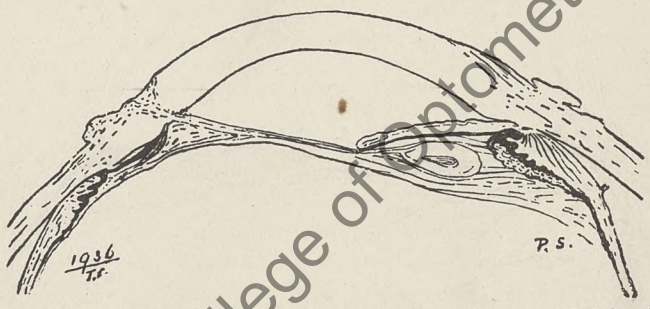
angle. A careful clinical examination usually reveals some entanglement of the iris or capsule in the scar, or some obstruction of the pupil by membrane or inflammatory exudation; but this is not always so, and the effect of such complications on the filtration-angle can seldom be made out in the living eye. In ten eyes blinded by this form of secondary glaucoma and

FIG. 10.

Secondary glaucoma after extraction of senile cataract.—Upward incision with large iridectomy; anterior capsule partly removed with capsule forceps; wound reopened on tenth day by knock on eye; excised four months later, with T + 2; cornea lined by iris, organizing blood-clot, inflammatory exudation, and lens-matter; filtration-angle closed at both sides. (Specimen and notes from Mr. Treacher Collins.)

microscopically examined, the filtration-angle was closed in the neighborhood of the scar in every case; in seven or eight of the ten it was closed at the opposite side of the eye also, and probably throughout the whole circle; where it was not closed, it was blocked by exudation. In nine of the ten

FIG. 11.



Secondary glaucoma after extraction of senile cataract.—Operation a modified Graefe, uncomplicated, followed by slight iritis; vision good for twenty-one months, then rapid failure without much pain; T +; capsule adherent to cicatrix; filtration-angle closed at both sides. (Specimen and notes from Mr. Treacher Collins.)

eyes the capsule was adherent to the scar, and in the tenth, from which the lens had been removed in its capsule, the hyaloid was adherent in like manner.[1] The accompanying illustrations show how such entanglements may lead to traction on the ciliary processes and compression of the filtration-angle, even at the most distant parts of the circle. (Figs. 10, 11, 12.) For-

[1] Treacher Collins, Trans. Ophth. Soc. of United Kingdom, vol. x. p. 108, 1890.

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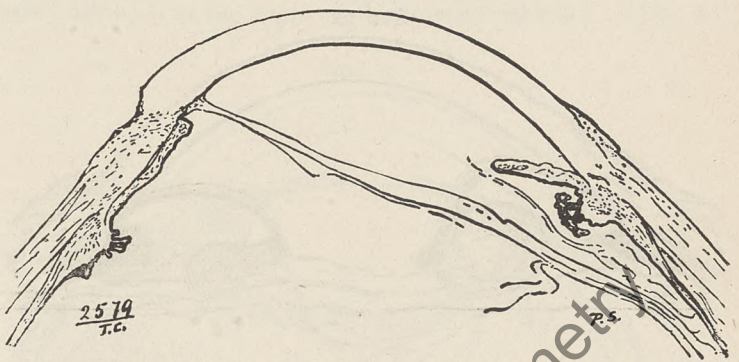
tunately,—for they are present more or less in a large number of well-operated eyes,—they do so only in a small minority of cases. When an eye which presents no definite complication of this kind, and which has enjoyed good sight for months after the operation, ultimately becomes glaucomatous, we may reasonably conjecture that a transparent membrane which at first gave no trouble has lately contracted, or that it has become less permeable than before, and has been pushed forward by an accumulation of fluid in the vitreous chamber. I have obtained proof of such an accumulation more than once when performing a sclerotomy for the relief of the high tension. On the withdrawal of the knife but little fluid escapes, and the iris at once applies itself closely to the cornea; on reintroducing the knife through the same wound and passing it through the pupil into the vitreous, a gush of fluid occurs; the iris retires, and the eye is at once slack,—a proof that the fluid was imprisoned behind a pupillary membrane.

FIG. 12.



Secondary glaucoma after extraction of cataract.—Result of operation good for twelve months, then pain, inflammation, and loss of vision; after excision, disk found to be deeply and widely excavated; iris and hyaloid adherent to cicatrix; filtration-angle closed at both sides. (Specimen and notes from Mr. Treacher Collins.)

In dealing with the glaucoma which follows cataract extraction, our aim should be to obtain an open pupil and an open filtration-angle. Sometimes a free division of the pupillary membrane with a cutting needle will suffice. More often a sclerotomy freely incising the angle of the chamber will be necessary; it should be combined with a free division of any pupillary membrane which may be present. A rare complication after cataract extraction is a total and persistent abolition of the anterior chamber, with high tension. A case of this kind within my knowledge was relieved by a scleral puncture combined with pressure on the cornea.

Needle operations and other simple wounds of the lens may induce glaucoma in various ways. For example, during a preliminary iridectomy for senile cataract the knife touches the lens. Twenty-four hours later there is an acute glaucoma. The incision is healed; the wounded and swollen lens presses the iris against the cornea throughout a large part of the circle. It is extracted at once, and the eye recovers quickly, with normal tension

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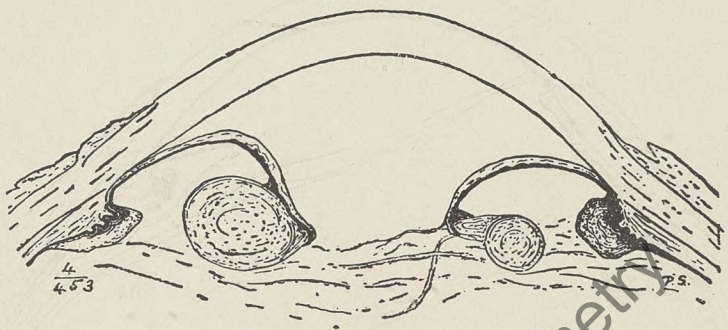
and good anterior chamber. In this and in the following example the sudden swelling of the lens is the cause of the glaucoma. In a boy with lamellar cataract, high tension sets in after the first needling. The lens appears to be swollen as a whole; there is no extrusion of its substance through the opening in the capsule. A second needling—without loss of aqueous—opens the capsule more widely; masses of lens-substance fall into the anterior chamber; the tension subsides, and the eye recovers without further drawback. In one instance, where the pupil was undilatable by atropine and the cataractous lens exceptionally small, a slight needling was followed at once by complete plugging of the pupil by the lens, with severe glaucoma; an iridectomy banished the glaucoma forthwith.[1] In other cases, again, high tension seems to depend on blocking of the angle of the anterior chamber by lens-substance and inflammatory exudation.

Again, a needle operation may lead to glaucoma through the occurrence of iritis and annular posterior synechia, the pupil-margin adhering to the

Fig. 13.



Secondary glaucoma after needle operation for cataract in infancy, seven years before excision.—Annular posterior synechia; accumulation of fluid behind iris; closure of filtration-angle.

capsule or hyaloid, and the iris bulging forward against the cornea, just as when the lens is present. (See page 633.) A timely iridectomy will, under favorable circumstances, relieve this condition and save a useful eye, while a long continuance of the glaucoma will lead to blindness with progressive distention of the eye, ultimately demanding excision. (Fig. 13.)

Dislocation of the Lens into the Anterior Chamber—A young adult, the subject of congenital malposition of both lenses, suffers no pain in the eyes until one day, while stooping over the wash-hand basin, she suddenly feels something wrong in one eye. Within an hour an intense glaucoma is established; the lens lies in the anterior chamber; the periphery of the iris is visibly in contact with the cornea; the rest of the iris bends sharply backward round the edge of the lens. Before extraction can be performed, the lens slips back through the pupil, and the glaucoma subsides. The dislocation recurs several times, and each time with glaucoma; ultimately the lens is extracted and the glaucoma appears no more. In another example,

[1] Communicated to the author by Mr. Lloyd Owen.

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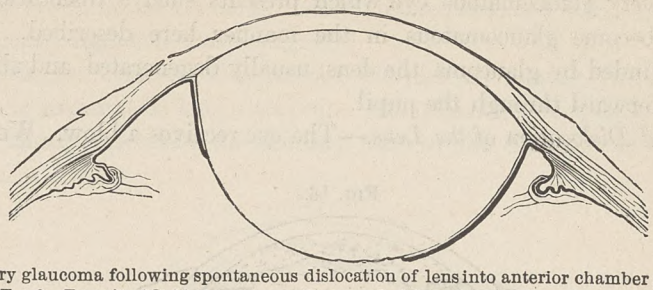
an eye already blind through pigmentary retinitis suffers a similar spontaneous dislocation of the lens, followed rapidly by an intense glaucoma.

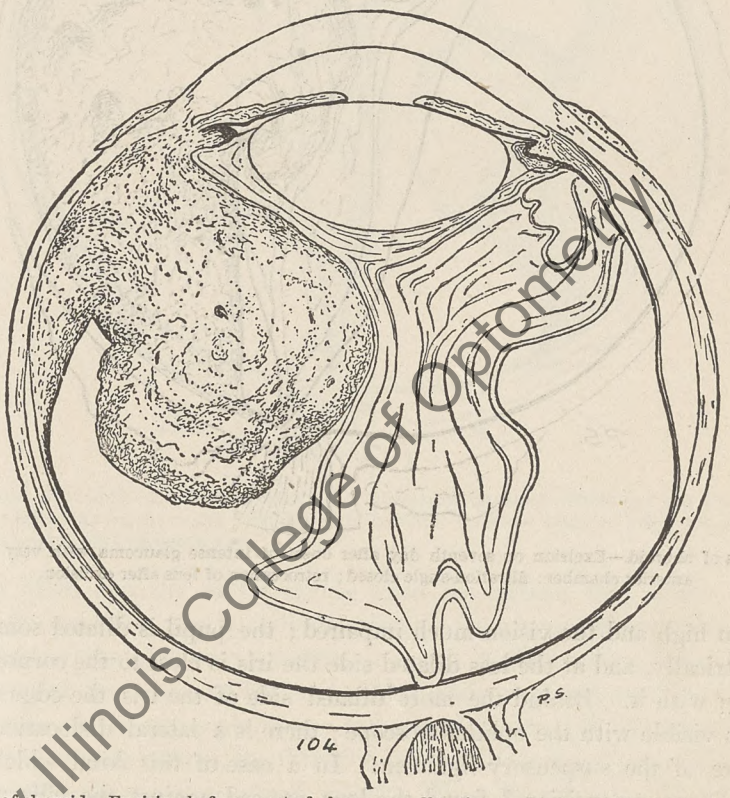
FIG. 14.



Secondary glaucoma following spontaneous dislocation of lens into anterior chamber; excision on ninth day. T + 3. Transit of fluid through pupil arrested; iris and lens driven forward against cornea and moulded to its curvature by pressure of retained fluid; access to filtration-angle cut off. (See Ophthalmic Review, 1882, p. 209.)

It is excised on the ninth day, and reveals in a striking manner the evidence of arrested filtration at the angle of the anterior chamber. (Fig. 14.)

FIG. 15.



Sarcoma of choroid.—Excision before onset of glaucoma; filtration-angle open. (Mr. McHardy's case.)

When this displacement occurs without glaucomatous complication, we may be sure that the pupil is not completely occluded. One case is on

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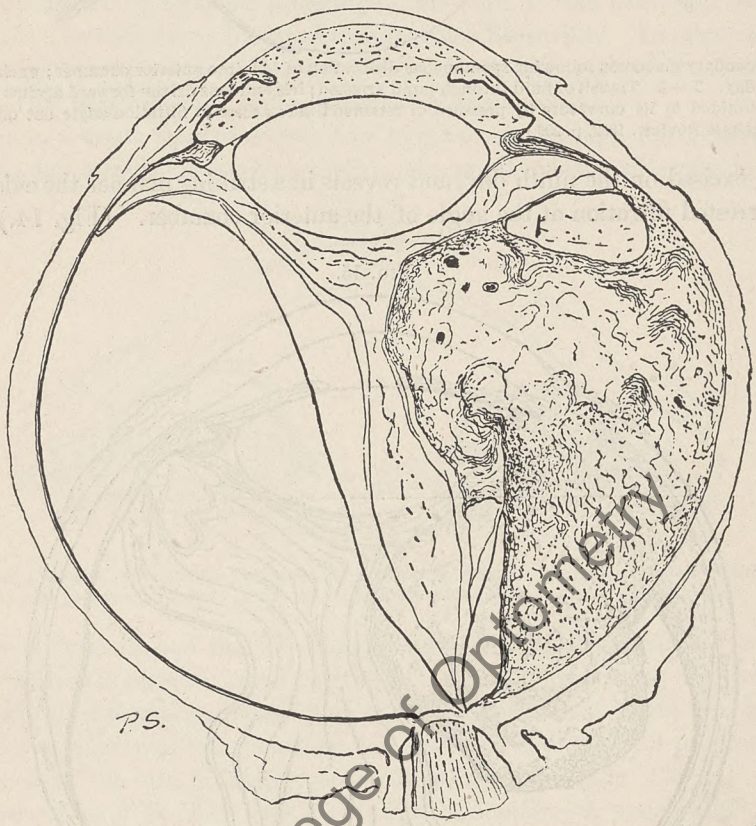
record in which there was no rise of tension until by the use of eserine the iris was contracted and tightened up against the posterior surface of the lens; an acute glaucoma then supervened.[1]

Not every glaucomatous eye which presents such a dislocation of the lens has become glaucomatous in the manner here described. In eyes already blinded by glaucoma the lens, usually degenerated and shrivelled, may fall forward through the pupil.

Lateral Dislocation of the Lens.—The eye receives a blow. We find the

FIG. 16.



Sarcoma of choroid.—Excision on seventh day after onset of intense glaucoma with very shallow anterior chamber; filtration-angle closed; retrocession of lens after excision.

tension high and the vision much impaired; the pupil is dilated somewhat excentrically, and at the less dilated side the iris is close to the cornea or in contact with it. Behind the more dilated side of the iris the edge of the lens is visible with the ophthalmoscope; there is a lateral dislocation with rupture of the suspensory ligament. In a case of this kind which ultimately came to excision I found the lens pressed against the ciliary body and iris at one side so firmly as to be indented by the ciliary processes,—

[1] J. L. Minor, New York Medical Journal, 1881, p. 194.

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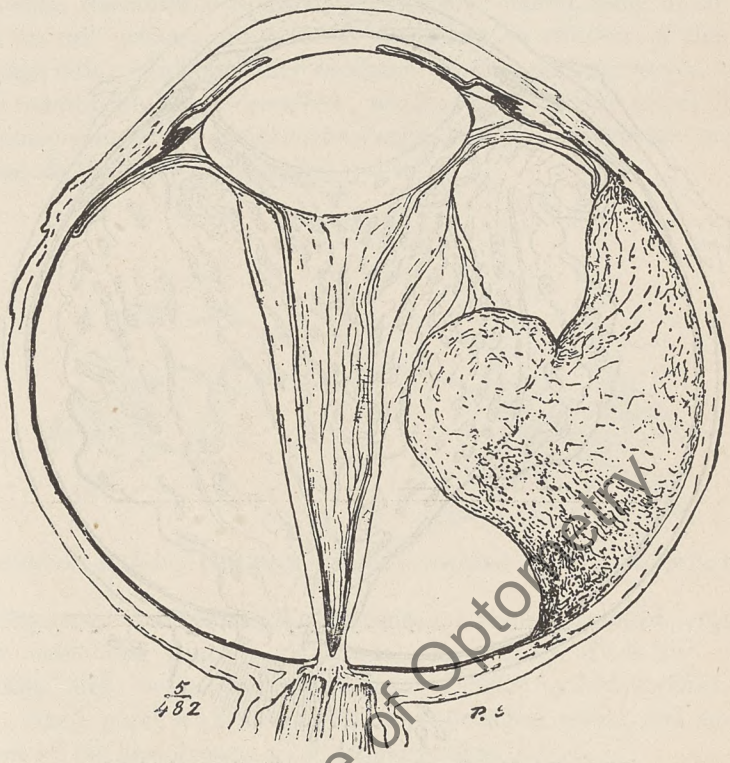
wedged in, so to speak, between the ciliary body in front and the consistent vitreous behind. The filtration-angle appeared to be closed throughout the whole circle; the vitreous body, displaced backward at the one side by the intrusion of the lens, was apparently driven forward at the other where the suspensory ligament was ruptured.

Intra-Ocular Tumors.—Sarcoma of the choroid almost always leads to glaucoma if excision be long delayed. The glaucoma is usually of acute type and closely resembles the primary form of the disease. If the eye be

FIG. 17.



Sarcoma of choroid.—Excision on sixth day of glaucomatous attack; filtration-angle closed; the angular bend in the iris shows the point to which the ciliary processes extended when the glaucoma was at its height. (Mr. Hodges's case.)

already blind and the media cloudy when the patient first comes under notice, the condition may be indistinguishable from acute primary glaucoma. The onset of the glaucoma coincides in point of time with a forward displacement of the lens and iris and with a consequent closure of the filtration-angle. If an eye containing a sarcoma of the choroid be excised while the tension is still normal, the filtration angle will be found open (Fig. 15); if during the glaucomatous attack, closed. (Figs. 16 and 17.) The advance of the lens is not due to direct pressure by the tumor, for in many cases the tumor is far removed from contact with the lens. (Fig. 17.) The process appears to be as follows. The growth of the tumor causes, probably by

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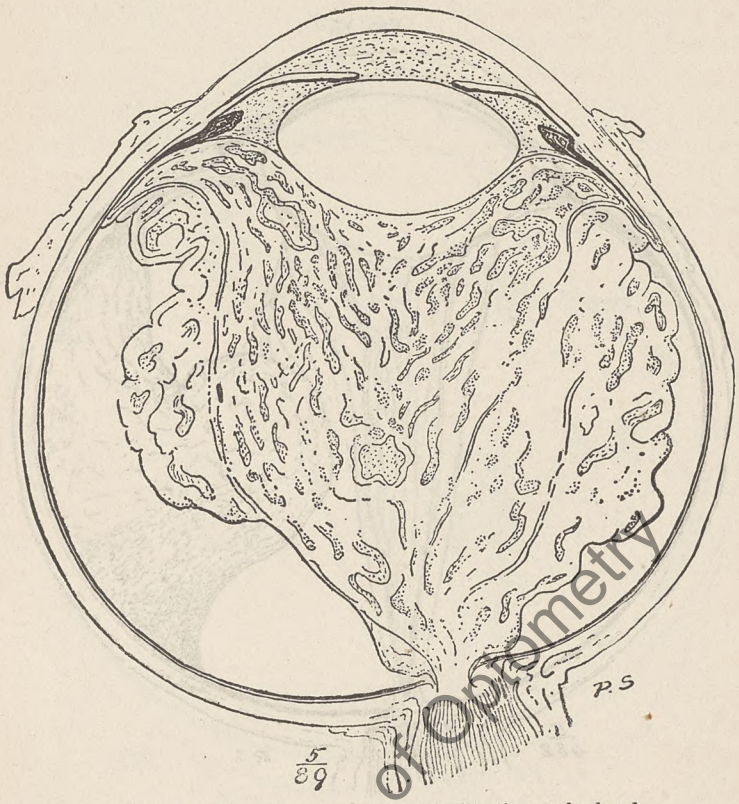
obstruction of the choroidal veins,[1] an outpouring of serum from the choroid; the retina is detached thereby and driven inward upon the vitreous. At first there is no discoverable rise of tension, for the vitreous makes room for the intrusion by parting with some of its fluid through the hyaloid into the aqueous chamber and so out of the eye. (See page 630.) Later, when the vitreous fluid is nearly all gone and the retina is folded together in the axis of the eye, further compensation becomes impossible; then the lens,

Fig. 18.



Glioma of retina.—Secondary glaucoma; filtration-angle closed.

ciliary processes, and iris are driven forward by the subretinal dropsical effusion, the filtration-angle is compressed, and the glaucoma begins.

The extreme forward displacement of the lens which is commonly present when the eye is excised has often disappeared when it is opened for examination after the necessary hardening process; the slackening of the globe which occurs after excision permits the lens to retire to its normal position.

Glioma of the retina usually leads in like manner to an advance of the lens with closure of the filtration-angle and a simultaneous onset of high tension. (Fig. 18.) The glaucoma is commonly of less violent type than

[1] Fuchs, Das Sarcom des Uveal Tractus, Vienna, 1882, S. 210.

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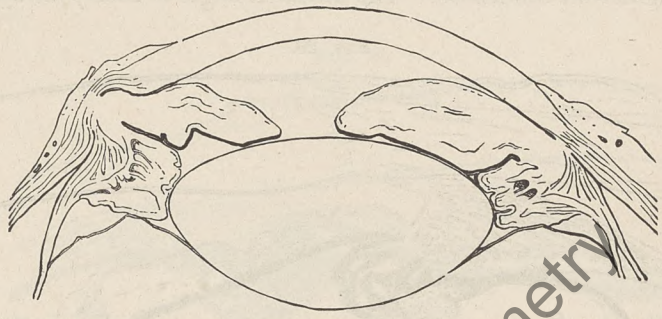
that induced by choroidal tumors, probably because the choroidal circulation is not directly obstructed.

Tumors of the iris occasionally induce glaucoma by direct blocking of the filtration-angle. (Fig. 19.)

Inflammatory and Serous Exudations.—Inflammatory exudation into the aqueous chamber as a cause of high tension has already been referred to. (Page 635.) Exudations into the vitreous chamber are also sometimes followed by high tension. In such cases the depth of the anterior chamber, if at all affected, is diminished rather than increased, and the condition may bear a close resemblance to primary glaucoma; indeed, some of the cases which we call primary are probably dependent on effusions of this kind. (See page 649.) Inflammatory exudation plays a part, moreover, in most of the conditions already described: whether a part of the initial disorder or a consequence of the glaucomatous complication, it aggravates and perpetuates the difficulty of filtration.

FIG. 19.



Tumor of iris and ciliary body blocking filtration-angle.—Secondary glaucoma. (Mr. McHardy's case.)

Intra-ocular hemorrhage is sometimes the starting-point of a condition closely resembling primary glaucoma. (Page 649.) It is also a cause of sudden high tension in eyes previously blinded by irido-cyclitis,—eyes with a closed pupil, a shrunken vitreous, a detached retina, and, until the moment of the hemorrhage, a subnormal tension.

Coloboma and Aniridia.—The glaucoma occasionally met with in eyes which present a partial or total absence of the iris is not, strictly speaking, secondary, but may conveniently be mentioned here. It forms no exception to the rule that high tension depends on a blocked filtration-angle. The iris in such eyes is represented by a rudimentary nodule, which is not visible through the cornea, but which when pushed forward blocks the filtration-area and adheres to it, as does the base of the normal iris in other cases. Moreover, in an eye blinded by high tension after the evulsion of the whole iris by an injury, the tips of the ciliary processes were found adherent in the same situation.[1]

[1] Treacher Collins, Ophth. Review, 1891, p. 101; and Trans. Ophth. Soc. of United Kingdom, 1893, vol. xiii. p. 134.

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Congenital Buphthalmos.—The glaucoma sometimes present at birth has been found to depend on absence of the filtration-angle. The peripheral adhesion of iris and cornea is here probably a fault of development—failure in the separation of these structures—rather than a product of disease.[1] In these eyes, though the angle is closed, the rest of the anterior chamber is generally deepened; the fluid accumulates in the aqueous, not in the vitreous, chamber. This shows that the primary obstruction is at the outlet of the aqueous chamber. The enlargement of the eye is the result of the increased pressure within.

Other disorders of the eye than those mentioned above may be met with in association with glaucoma; they do not necessarily stand to it, in all cases, in the relation of cause to effect.

CAUSES OF PRIMARY GLAUCOMA.

The causes of primary glaucoma are more difficult to trace than those of the forms already described. In eyes blinded by the disease we find the following significant conditions. The filtration-angle is nearly always closed;

FIG. 20.

From the healthy emmetropic eye of a man aged fifty-seven; for comparison with Figs. 21 to 24.

FIG. 21.

From an eye blinded by primary glaucoma; acute; recent.—Ciliary processes swollen and advanced; iris-base pressed against cornea, but not adherent to it.

when not closed it usually shows signs of compression. In recent cases the iris-base is merely pressed against the cornea and ligamentum pectinatum (Fig. 21); in older cases it is generally adherent (Figs. 22, 23, and 24), and

[1] Treacher Collins, Hunterian Lectures, Lancet, December, 1894.

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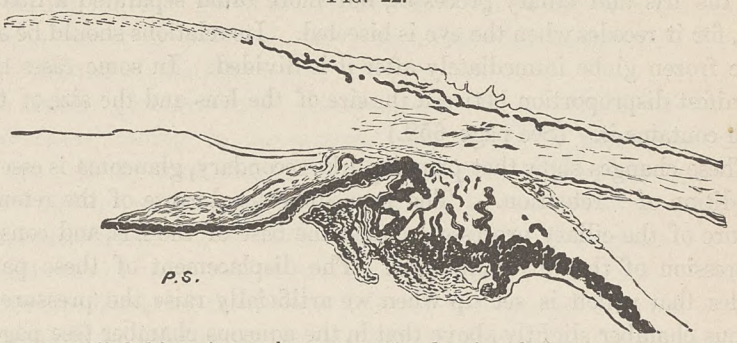
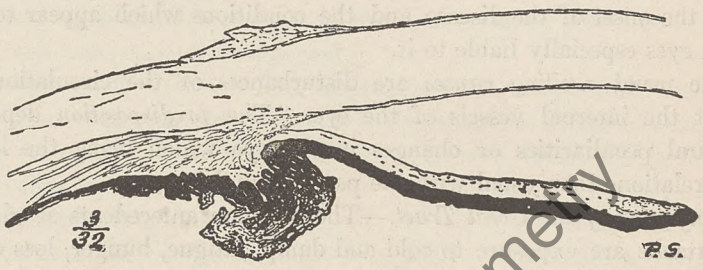
often much atrophied. The adhesion varies in extent in different eyes and in different parts of the same eye, being sometimes one millimetre or more in width, sometimes so slight as to be easily overlooked. (Fig. 23.) The ciliary processes in recent cases are enlarged and advanced, and show signs

FIG. 22.



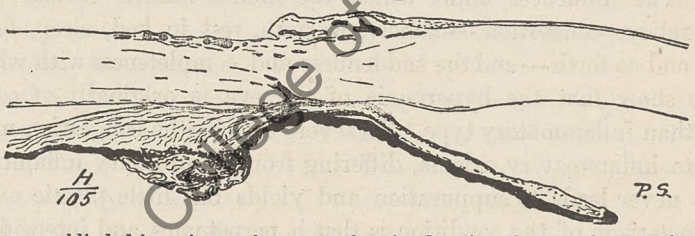
From an eye blinded by primary glaucoma; acute; duration about twelve months.—Filtration-angle closed by adhesion; ciliary processes moulded into a wedge-like form by compression between lens and iris.

FIG. 23.



From an eye blinded by primary glaucoma; chronic; duration about five years.—Iris-base and ligamentum pectinatum adherent only to a very small extent; a very much wider adhesion exists at the opposite side of the eye.

FIG. 24.



From an eye blinded by primary glaucoma; chronic; duration about twelve months.—Iris-base adherent to cornea; ciliary processes atrophied. The eye went blind gradually, without pain or redness; blind for five months; pain and redness for three weeks before excision.

of having been squeezed between the iris and the lens. (Fig. 22.) Viewed in transverse section, they present an increase of thickness also, the interspaces being narrowed or partly obliterated. In cases of long standing they are usually atrophied and retracted (Fig. 24), but have often left an impress in the base of the iris in evidence of their previous turgescence and advance-

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ment. Between extreme hypertrophy and extreme atrophy there may be any degree of enlargement or shrinking. The ciliary muscle shares in these changes; at first it is drawn forward with the ciliary processes, later it retracts and atrophies. The lens usually lies nearer to the cornea than in normal eyes at the same period of life. It is sometimes in close contact with the iris and ciliary processes, but more often separated a little from them, for it recedes when the eye is bisected. Its relations should be studied in the frozen globe immediately after it is divided. In some cases there is a manifest disproportion between the size of the lens and the size of the eye which contains it. (See page 652.)

These changes show that primary, like secondary, glaucoma is essentially a condition of "retention." The immediate local cause of the retention is pressure of the ciliary processes against the base of the iris, and consequent compression of the filtration-angle. The displacement of these parts resembles that which is set up when we artificially raise the pressure in the vitreous chamber slightly above that in the aqueous chamber (see page 632), and is almost identical with that which is induced by the growth of a choroidal tumor; but there is no gross lesion to account for it. The initial causes can only be discovered by closely observing the circumstances which attend the onset of the disease and the conditions which appear to render certain eyes especially liable to it.

The usual *exciting causes* are disturbances of the circulation which congest the internal vessels of the eye. The *predisposition* depends on structural peculiarities or changes in the eye which bring the lens into closer relations than usual with the parts around it.

Congestion of the Uveal Tract.—The common antecedents of glaucomatous attacks are exposure to cold and damp, fatigue, hunger, loss of sleep, depressing emotion, constipation, hepatic derangement, heart-weakness, bronchitis,—in short, various conditions which disturb the circulation and congest the venous system. The congestion is often manifest in the temporal veins. The influences under which the milder attacks subside are those which relieve congestion,—namely, warmth, rest in bed, sleep, food, purgation, and so forth,—and the suddenness and completeness with which they subside show that the hyperæmia of the eye is originally of congestive rather than inflammatory type. In severe attacks the congestion mounts to an acute inflammatory œdema, differing from an ordinary inflammation in that it never leads to suppuration and yields but little plastic exudation. A characteristic of the condition is that it perpetuates and intensifies itself in a vicious circle. The turgid ciliary processes press against the base of the iris and narrow the filtration-angle; the escape of fluid is retarded, and the intra-ocular pressure rises; the increasing pressure hinders the flow of blood through the choroidal veins and augments the swelling of the processes; this, in its turn, increases the compression of the filtration-angle. A typical acute glaucoma is an inflammatory disease in the same sense that a strangulated hernia is so, but not otherwise; it exhibits an acute obstruc-

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tion of the circulation which can be cut short by removal of the pressure, and in no other way.

The initial disturbance of the circulation may arise from local as well as from systemic causes,—*e.g.*, from contusion of the head, from contusion of the eye without discoverable lesion, or from a trivial burn or abrasion of the cornea. When glaucoma follows such an injury, a predisposition to it may generally be assumed, and is sometimes demonstrated by the subsequent behavior of the fellow-eye.

In a certain group of cases glaucoma depends on *disease of the blood or blood-vessels* leading to thrombosis, hemorrhage, or morbid exudation in the eye. Such cases should be distinguished, if possible, from those which are strictly primary, for their treatment will otherwise be apt to end in unexpected failure. The distinction is often difficult to make, however, for the glaucoma closely resembles a primary attack; the excess of fluid is in the vitreous chamber, the lens is more or less pushed forward, and the filtration-angle is compressed by the ciliary processes. (The suggestion that primary glaucoma depends on occlusion of the vortex veins[1] is certainly not correct for the generality of cases. Thirteen glaucomatous eyes, microscopically examined by me and compared with six non-glaucomatous eyes, showed no distinctive changes in the vortex veins;[2] and this observation has been independently confirmed.[3])

Dilatation of the Pupil.—The application of atropine, homatropine, cocaine, or any other mydriatic is apt to aggravate an incipient glaucoma, and may even light up a severe attack in an eye which has previously shown no sign of the disease. When the filtration-angle is already narrow, the peripheral folding and thickening of the iris which occur with dilatation of the pupil may suffice to block it entirely. Fortunately, the timely use of a myotic will often undo the mischief.

Predisposition.—The causes enumerated above are obviously insufficient by themselves to explain the occurrence of glaucoma, for they are present in innumerable persons with no such result. When they induce glaucoma, they generally act in conjunction with the predisposing influences described in the following paragraphs.

Influence of Age and Sex.—The following facts were deduced from the systematic tabulation of one thousand cases of primary glaucoma observed by ophthalmic surgeons in the United Kingdom,[4] the figures representing the frequency of the disease as actually met with being adjusted by means of life tables to the numbers of persons, male and female, living in each period of life, so as to show the liability belonging to each life-period and

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[1] Birnbacher and Czermak, Von Graefe's Archiv, xxxii., ii., S. 1.

[2] Author's Lectures, p. 129.

[3] Stirling, Roy. Lond. Ophth. Hosp. Reports, vol. xiii., Part IV., p. 421.

[4] Trans. Ophth. Soc. of United Kingdom, vol. vi. p. 294, 1886. See also statistics by Knapp, Trans. Amer. Ophth. Soc., 1889; by Neuburger, Centralblatt f. Augenheilk., 1894, S. 13; and by Zentmayer and Posey, Arch. of Ophth., xxiv. 3, 1895.

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each sex. (See Chart, Fig. 25.) No other statistics, equally systematic, have hitherto been collected on so large a scale.

The liability to primary glaucoma is extremely slight in childhood and youth. It steadily increases throughout life up to and during the seventh decade,—*i.e.*, the ten years from sixty to seventy,—and probably to a still greater age, for there is evidence to show that our registers do not adequately represent the relative frequency of the disease in the very old, who, for various reasons, fail to present themselves for treatment. At sixty-five it is at least one hundred times greater than at fifteen, and more than twice as great as at forty-five years of age.

The liability of females is greater than that of males in a ratio probably of about six to five. This extra liability pertains to the whole of life, except, perhaps, the periods before thirty and after seventy, concerning which the data are too few to justify generalization. It relates markedly to the acute congestive forms of the disease, not to the non-congestive. The greater instability of the vaso-motor system in women, and especially the vascular disturbances connected with the generative system, may explain this difference.

The continuous increase of liability throughout life appears to depend mainly on the continuous growth of the crystalline lens. The cornea attains its maximum diameter as early as the fifth year; the globe is fully grown at the beginning of adult life, if not earlier; but the lens, so long as it remains healthy, continues to grow from youth to old age. During the forty years between twenty-five and sixty-five it adds about one-tenth to its diameters and one-third to its volume.[1] Consequently, as age advances, it encroaches more and more on the space in which it lies, its margin coming into closer relation with the ciliary processes and its anterior surface approaching nearer to the cornea. These physiological changes are, as a rule, completely compatible with the integrity of the eye, but they sometimes pass the limit of safety. If the ciliary processes have not sufficient space at their disposal, they are apt, especially during periods of turgescence, to press against the iris-base and compress the filtration-angle; when the anterior chamber becomes unduly shallow, the iris, especially when thickened by dilatation of the pupil, is apt to block the outlet. In this way the narrowing of the circumlental space and the shallowing of the anterior chamber which accompany the advance of life involve an increasing liability to glaucoma.

With the advance of life, moreover, the tension of the zonula diminishes. As the lens gains in size and loses elasticity the zonula fibres have a narrower gap to bridge and a lessened traction from the lens-capsule. When a young eye is bisected, the tense zonula at once reacts on the contour of the globe, dragging the ciliary body inward and increasing the curvature of the cornea; when an old eye is similarly treated, there is much less of

[1] Trans. Ophth. Soc. of United Kingdom, 1883, p. 79.

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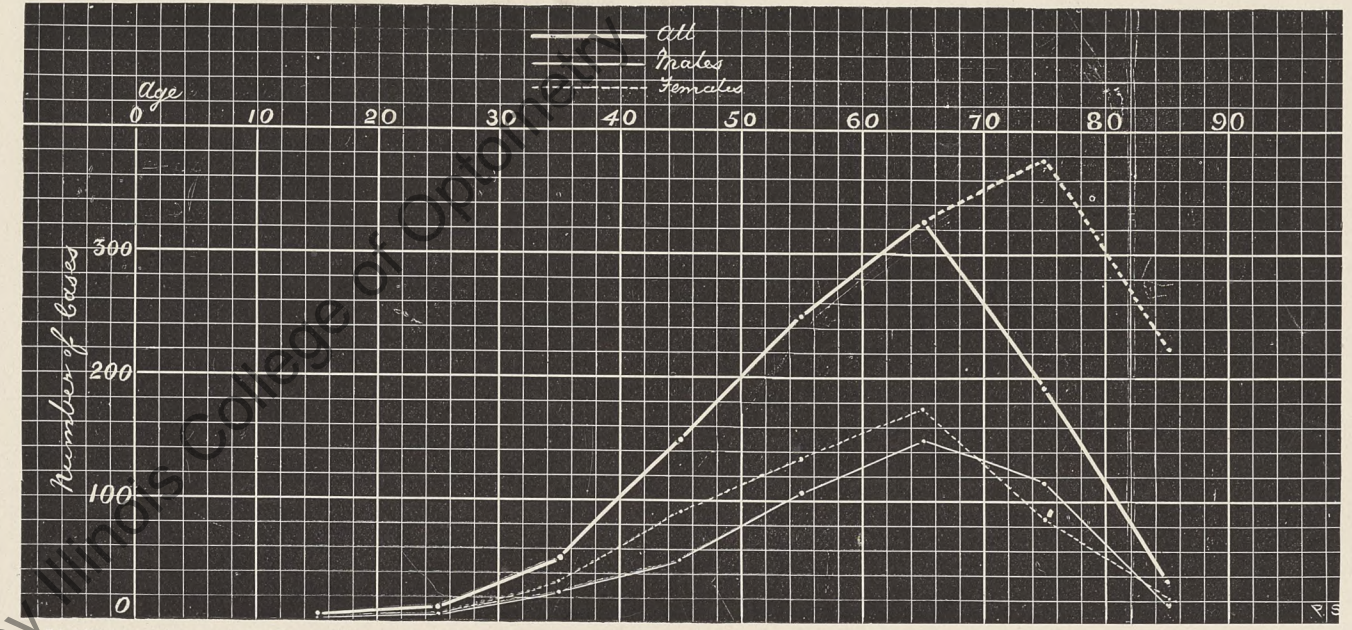
FIG. 25.



Liability to primary glaucoma at different periods of life, deduced from one thousand actual cases redistributed as though persons of all ages and both sexes were equally numerous.

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this displacement, for the zonula is comparatively slack. The loss of tension in the zonula diminishes the stability of the lens and renders it more liable to displacement forward.[1] In some eyes blinded by glaucoma the tension of the zonula seems to be entirely lost, and sometimes after a successful iridectomy the long-continued shallowness of the anterior chamber shows that it is very low.

The shallow anterior chamber common in primary glaucoma is not necessarily due to displacement of the lens, for in old age the chamber is normally shallow by reason of the greater thickness of the lens; but in many cases, especially of the acute kind, the lens is certainly displaced, for when the attack passes off the chamber deepens. This indicates an accumulation of fluid in the vitreous chamber. In the normal eye any excess of fluid in the vitreous can readily escape into the aqueous chamber (see page 630), and during the growth of choroidal tumors, as we have seen (page 644), much of the vitreous fluid escapes in this way before the lens becomes displaced. It would seem, therefore, that in eyes suffering from primary glaucoma there is frequently some special condition which hinders the transit of fluid from the vitreous to the aqueous chamber. Changes in the hyaloid membrane, in the vitreous tissue, or in the fluid itself are possible impediments to filtration, and in eyes blinded by glaucoma we often find the hyaloid and the septa of the vitreous thickened or coated by albuminous coagula. In many cases, however, especially of acute glaucoma, the accumulation of fluid behind the lens seems to be due rather to obstruction of the circumlental space by the swollen ciliary processes. The turgid processes are compressed between the lens and the iris, and the fluid which they secrete into the vitreous is unable to find an exit thence.

In addition to the physiological changes above described, the advance of life brings with it an increasing tendency to the vascular and other degenerative changes which are more or less concerned in the causation of glaucoma. These, therefore, are partly responsible for the greater liability of the old.

Smallness of the Eye.—Small eyes are especially liable to primary glaucoma. They may be recognized during life by the smallness of the cornea. They are not, be it observed, the only eyes which suffer, for the disease is met with in eyes of average and of more than average size; but they suffer with a frequency which is disproportionate to their number. Eyes in which the cornea measures only ten millimetres in the horizontal diameter seem seldom to escape the disease. Fortunately, they are rare. My observations on this point are sufficiently numerous and precise to justify a positive statement, so far as English people are concerned.[2] In order to establish a standard for comparison, the horizontal diameter of the cornea was measured,

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[1] Snellen, Trans. Internat. Ophthal. Congress at Heidelberg, 1888, p. 249; also Ophthalmic Review, February, 1891, p. 47.

[2] Trans. Ophth. Soc. of United Kingdom, 1890, vol. x. p. 68.

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by means of a simple keratometer designed for the purpose, in one thousand healthy eyes belonging in equal number to the two sexes and representing all periods of life from five to ninety years of age. The refraction was noted in every case. Similar measurements were made in one hundred and twelve persons—fifty-one males and sixty-one females—suffering in one eye or in both from primary glaucoma. Their average age was fifty-seven. The number of eyes was two hundred and sixteen, some of the patients having only one, or only one which could be measured. Of these two hundred and sixteen eyes, one hundred and sixty-nine were glaucomatous, and forty-seven healthy. The average horizontal diameter of the cornea in the thousand healthy eyes was 11.6 millimetres; in the glaucomatous, including unaffected fellow-eyes, it was 11.17 millimetres. To put the point in another way, small corneas were much commoner in the glaucoma group than in the healthy group. Thus, a diameter of 10.5 millimetres was found in 22.7 per cent. of the former against 1.70 per cent. of the latter. More significant still, a diameter of 10 millimetres was found in nine of the two hundred and sixteen glaucomatous eyes (4.17 per cent.), but not once in the one thousand healthy eyes. In some of the glaucoma patients, when first examined, both corneas were small, but only one eye glaucomatous, and in several of these cases glaucoma attacked the fellow eye later, a proof that the smallness of the cornea precedes the glaucoma and is not caused by it.

In the next place, it was ascertained by means of complete measurements of a large number of eyeballs that a small cornea indicates a small globe. Lastly, eighteen eyes blinded by primary glaucoma were completely measured and compared with twenty-five healthy eyes.[1] The average dimensions in the two groups respectively were as follows:

	CORNEA.		GLOBE.		
	Horizontal.	Vertical.	Horizontal.	Vertical.	(Antero-Posterior.)
Healthy	11.6	11.0	24.4	24.0	(24.8)
Glaucomatous . .	11.0	10.3	23.3	22.8	(23.3)

The fact thus established is, be it again observed, not that primary glaucoma is a disease of small eyes only, but that small eyes are especially liable to it. They appear, moreover, so far as present evidence goes, to be attacked earlier in life than others.

The greater liability of small eyes appears to depend on their containing disproportionately large lenses. This disproportion is most obvious in the extreme cases commonly known as microphthalmos. In such eyes the lens, unless it be degenerated, is, as a rule, too large for the globe.[2] In eyes of less pronounced smallness the disproportion is less obvious, but is mani-

[1] Author's Lectures, p. 106.

[2] See cases reported by Hocquard and Masson, Archives d'Ophtalmologie, 1883, p. 231; by Lang and Treacher Collins, Roy. Lond. Ophth. Hosp. Reports, vol. xii. p. 292; and by Kundrat, Ueber die angeborenen Cysten im unteren Augenlide, Mikrophthalmie und Anophthalmie, Bergmann, Vienna.

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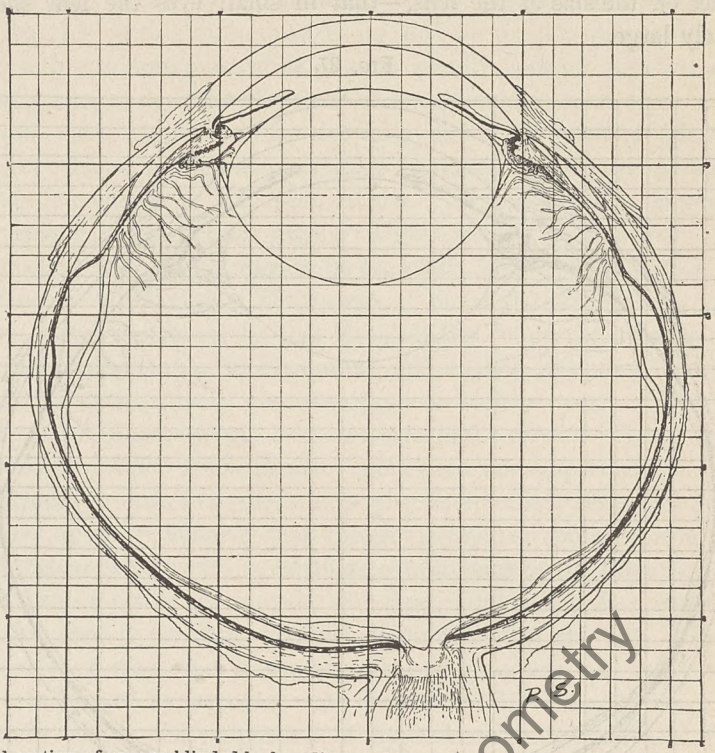
fest enough when accurate comparison is made with larger eyes belonging to the same life-period. The drawings here given (Figs. 26 and 27) of a small eye blinded by hereditary glaucoma and a full-sized healthy eye of nearly the same age exhibit a striking difference in the relative size of the

FIG. 26.



Horizontal section of an eye blinded by hereditary primary glaucoma. Age of patient, twenty-nine. The divisions represent millimetres.

lenses, that of the small eye being actually somewhat larger than that of the large one. The dimensions in millimetres may be here compared:

	GLOBE.			CORNEA.		LENS.	
	Horiz.	Vert.	Ant.-Post.	Horiz.	Vert.	Horiz.	Ant.-Post.
Glaucomatous eye . .	22	21	21.75	10	10	9	6.5
Healthy eye	25	24	25	12	11.5	8.75	6

Evidently the growth of the lens is not much influenced by variations of size in the parts around it. The reason is not far to seek. The lens springs from the ectoderm of the embryo and severs its connection with the parent membrane early; it remains an isolated mass of epithelium which, unlike that of any other part, proliferates within a closed capsule and has no free surface. Before the end of fœtal life it loses all vascular connection with the rest of the eye, and is freed from all extrinsic resistance to its growth, except the pressure of the fluid which surrounds it and the traction of the suspensory ligament. It is as unique in its growth as in its

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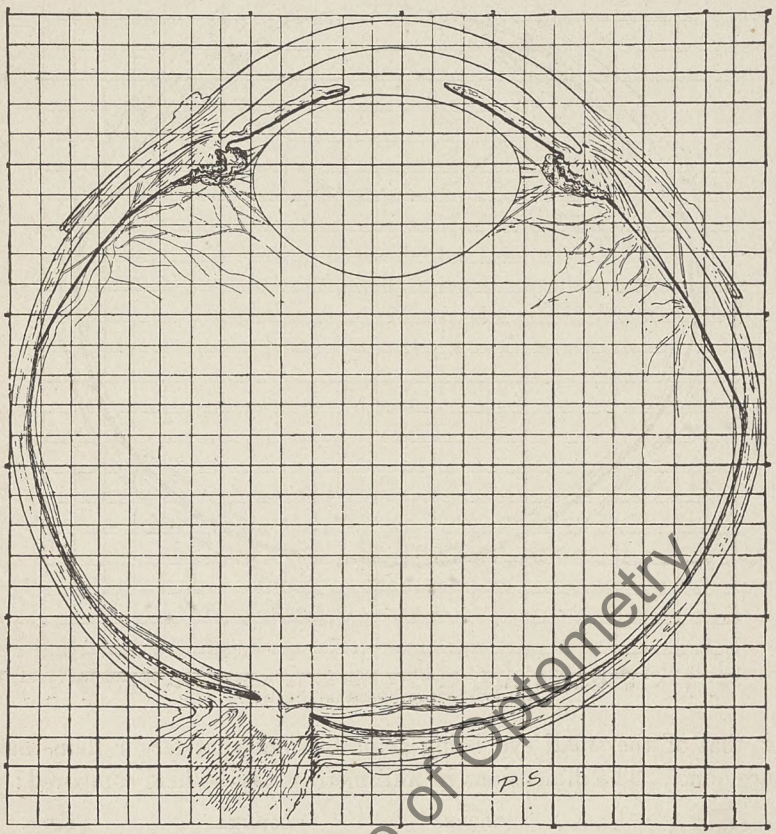
structure and its relations: it enlarges continuously throughout life. On the other hand, the structures which determine the size of the eyeball—the cornea and sclera—spring from the mesoderm, making their appearance later and completing their growth much earlier. It is therefore not surprising that maldevelopment of the cornea and sclera should have little influence on the size of the lens,—that in small eyes the lens should be relatively large.

Fig. 27.



Horizontal section of a healthy emmetropic eye removed, together with an orbital tumor, during life, from a man aged thirty-five. The divisions represent millimetres.

Heredity has been traced in many cases,—*e.g.*, in a mother and two daughters, in a father and three sons, in a mother and three sons, and in one family through several generations. Various more or less plausible explanations have been offered,—viz., the inheritance of gout, of vascular disease, of nerve-irritability, or of rigidity of the sclera,—but they are not based on very cogent evidence. The only case of hereditary glaucoma which I have been able to investigate was associated with well-marked hereditary smallness of the eyes. It involved a father and daughter. The father lost both eyes by congestive primary glaucoma, the right at fifty-two, the left at forty-two years of age; the cornea of each measured 10.5 millimetres

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horizontally. The daughter was attacked in both eyes at the age of twenty-nine. The right was excised when blind by another surgeon, who kindly placed it at my disposal; the left was treated by scleral puncture and iridectomy, and recovered useful sight. Both corneas measured ten millimetres horizontally; the excised eye was exceptionally small and contained a relatively large lens.[1] (Figs. 26 and 27.) Whether hereditary glaucoma usually depends on this cause must be decided by further observation; but it is interesting to note meanwhile that it usually declares itself at a relatively early age, as does the glaucoma of exceptionally small eyes.

Certain races appear to be specially liable to primary glaucoma. Brugsch-Bey found a higher percentage of cases among eye patients at Cairo than is common in European clinics.[2] Moura found a larger percentage among negroes than among whites at Rio de Janeiro.[3] Some writers declare the Jewish race to be especially liable; but on this point the evidence is conflicting. The causes of racial liability are not yet determined; but it is noteworthy that Brugsch-Bey found the average cornea smaller in Egyptians than in Europeans, and among Egyptians smaller in eyes suffering from primary glaucoma than in healthy eyes.

Influence of Hypermetropia and Accommodative Strain.—A majority of the eyes which suffer from primary glaucoma are hypermetropic. The hypermetropic eye has, as a rule, a more prominent ciliary body and perhaps a shallower anterior chamber than the emmetropic or the myopic eye, and these conditions would tend to facilitate compression of the filtration-angle. Moreover, an excessive accommodative effort tends to slacken the zonula unduly and thus to facilitate forward displacement of the lens. But the influence of hypermetropia in the causation of glaucoma has not yet been very clearly proved by statistics. At the time of life when primary glaucoma chiefly occurs the proportion of hypermetropes is increased by the acquired hypermetropia of old age, and it has not yet been shown that hypermetropia is more prevalent among glaucoma patients than among the general population in the same life-periods.[4] The special liability of small eyes does not prove a special relation to hypermetropia, for small eyes are

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[1] For details of this case and references to other examples of hereditary glaucoma, see Ophth. Review, 1894, p. 215.

[2] Trans. Internat. Medical Congress, Washington, vol. iii. p. 752, 1887.

[3] The same, p. 755.

[4] Kryoukoff, in Moscow, found the following refractive conditions in cases of primary glaucoma: hypermetropia, 43.18 per cent.; emmetropia, 28 per cent.; myopia, 28.78 per cent. Among more than ten thousand consecutive ophthalmic patients tested with regard to refraction, he found hypermetropia, 43.09 per cent.; emmetropia, 27.58 per cent.; myopia, 29.32 per cent. He therefore concludes that the frequency of hypermetropia in glaucoma is not relatively greater than its frequency in general. (Abstract in Ophthalmic Review, vol. viii. p. 370. See also note by G. A. Berry, vol. ix. p. 58.) But this conclusion is open to the objection that ophthalmic cases in general probably present a higher proportion of hypermetropia than does the general population, and therefore afford no fair standard for a comparison of this kind.

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not necessarily hypermetropic, and hypermetropic eyes are not necessarily small. On measuring the cornea in ninety eyes with high hypermetropia and ninety with high myopia, I found the averages exactly equal. I found, moreover, that of eyes with small corneas about half were hypermetropic, the other half emmetropic or myopic.[1] It seems, therefore, that it is rather the small eye, as such, than the hypermetropic eye that is especially liable to primary glaucoma.

Summary of Causes.—The remote causes of glaucoma are very various. They include constitutional diseases, such as rheumatism, gout, syphilis, and many others; disorders of the respiratory, vascular, and nervous systems; injuries of many kinds; morbid growths; congenital imperfections; and senile changes.

The immediate cause in every case is an obstruction in the path of the intra-ocular fluid. The obstruction is different in different forms of the disease. It is usually concealed from inspection in the living eye, but its situation may commonly be inferred from the visible conditions.

When the anterior chamber is abnormally deep, the lens and iris being displaced backward, the obstruction lies either in the contents of the chamber or in the tissues which form the filtration-area.

When the iris is bulged forward by retention of fluid in the posterior aqueous chamber, the obstruction is primarily at the pupil, secondarily at the periphery of the chamber. In glaucoma following perforating injury or ulcer of the cornea, the displacement of the iris or lens usually indicates obstruction of the filtration-angle.

When the pupil is patent and the anterior chamber shallower, or at least not deeper, than in the healthy eye, we may usually diagnose compression of the filtration-angle by pressure of the ciliary processes against the base of the iris. The initial cause of the displacement is sometimes the intrusion of a tumor or an effusion of blood into the vitreous chamber: if we can exclude these factors we call the glaucoma primary. Primary glaucoma appears usually to depend on some vascular disturbance which congests the uveal tract, or upon a faulty relation of the lens to the parts around it, or upon both. If the patient be elderly, we know that the lens is relatively large. If the cornea be small, we may infer that the whole eyeball is small and that the relations of the lens are such as to predispose to compression of the filtration-angle, especially during dilatation of the pupil. An obstruction in the region of the hyaloid and the circumlental space, which checks the escape of surplus fluid from the vitreous and leads to an advance of the lens, appears to be present in many cases. Slackness of the zonula, with consequent instability of the lens, is probably a contributory cause. Through one or other of these causes, or several in combination, the ciliary processes are pressed against the iris, and the filtration-angle is narrowed or closed.

[1] Trans. Ophth. Soc. of United Kingdom, vol. x. pp. 72 and 75.

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SYMPTOMS AND COURSE.

The symptoms of glaucoma are numerous and somewhat complex, and they vary greatly in different forms of the disease, but they are readily intelligible when referred to their essential cause, the excess of pressure within the eye. They are the expression for the most part of over-tension of the tunics, embarrassed circulation, and impaired nutrition, and it will be well to review them with special regard to their causation before describing the chief clinical types of the disease. The following paragraphs refer chiefly to the symptoms of primary glaucoma; those of secondary glaucoma are essentially the same, but are often masked or modified by the coexisting disease.

Increased tension of the eye is the leading symptom of glaucoma. It is detected as follows. The patient looks downward without closing the eyes tightly. The surgeon, standing in front and steadying his hands by resting the outer fingers of each on the patient's forehead, places the tips of his two index fingers on the upper eyelid, and with gentle alternate pressure feels the globe behind the corneal region. He then feels the fellow-eye in like manner for comparison. This test should be in constant use in dealing with eye disorders. It will reveal an excess of tension in many eyes which are outwardly healthy in appearance, and normal tension in many which, according to the patient, suffer from a sense of fulness.

Degrees of tension are commonly described and recorded by means of Bowman's symbols,[1] which, in the slightly altered form now usually employed, are as follows:

Tn: Tension normal.
T + 1?: Doubtful increase of tension.
T + 1: Slight but positive increase of tension.
T + 2: Considerable tension; the finger can slightly impress the coats.
T + 3: Extreme tension; the finger cannot dimple the eye by firm pressure.
T — 1?: Doubtful reduction of tension.
T — 1: Slight but positive reduction of tension.
T — 2, T — 3: Successive degrees of reduced tension less easily defined by words.

For routine use the test above described is the best at our disposal, and is likely to remain so, but it is obviously inexact; for, though the sense of touch may be highly educated, we cannot measure or define what we feel. Many attempts have been made, therefore, to replace the finger by some form of mechanical tonometer. The mechanical method, like the digital, takes the resistance of the tunics as the index of the internal pressure, but aims at measuring it with precision. The following methods have been employed:

1. The instrument impresses the surface of the eye by means of a small rod or staff exerting a *given pressure*, and indicates the depth of

[1] Brit. Med. Journ., October 11, 1862.

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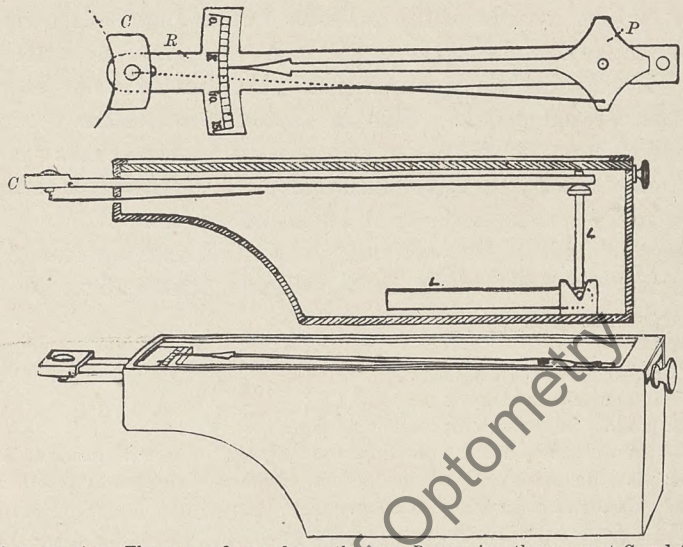
the impression produced.[1] 2. It impresses the surface of the eye in like manner but to a *given depth*, and indicates the amount of pressure employed.[2] 3. It flattens the surface of the eye by applying to it a plane surface exerting a *given pressure*, and indicates the size of the flattened area.[3] 4. It flattens a *given area* in like manner, and indicates the amount of pressure employed.[4]

Unfortunately, no instrument applied externally, however well designed, can accurately measure the pressure in the chambers, for the resistance of the tunics to external pressure varies not only with the internal pressure, but with the size and contour of the eye and with the elasticity and pliability of the tunics themselves. A well-constructed tonometer has, however, certain advantages over the finger. The instrument designed by the writer embodies the first of the methods above stated.[5] A small ivory

FIG. 28.



Author's tonometer.—The upper figure shows the ram *R*, carrying the crescent *C* and the pointer *P*, these two being connected by a wire,—seen from above. The second figure shows the instrument in section, the end of the ram resting on the upright arm of the rectangular lever *L*.

rod with rounded end presses on the eye with a force equal to a weight of ten grammes; a pointer and scale indicate the depth of the pit produced, in fractions of a millimetre. The instrument has no great advantage over the finger for comparing the eyes of one person with those of another, for

[1] Snellen and Landolt, Graefe-Saemisch Handbook, iii., S. 191; Priestley Smith, Ophth. Review, 1887, p. 33.

[2] Monnik and Weber, Graefe-Saemisch Handbook, iii. S. 189.

[3] Maklakoff, Arch. d'Ophth., vol. xii. p. 321.

[4] Fick, Inaugural Dissertation, Würzburg. 1888; and Transactions of International Ophthalmic Congress, Heidelberg, 1888, p. 289; also author's Lectures, p. 7; Koster, Von Graefe's Archiv, xli., ii., S. 130.

[5] Ophth. Review, 1887, p. 33.

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differences of tension which are so slight as to elude the sense of touch may be due to differences of curvature, etc., in the membranes. It is chiefly useful for comparing the two eyes of the same person, or for measuring the changes which occur in one and the same eye. Applied to the same spot of the same eye at different times, it reveals slight changes of tension with much greater certainty than the finger, and its indications, which are free from bias, can be recorded and compared. It is not an instrument for rough-and-ready diagnosis in unskilled hands, for it is more difficult to use than the finger test, but to those who would accurately control the indications afforded by their sense of touch it may be recommended.

The state of the tension of the eye, even though measured by the tonometer, will not always suffice to determine the presence or absence of glaucoma, for the increase is sometimes very slight and in many cases intermittent: the diagnosis must rest on a survey of all the symptoms.

Ciliary Injection.—In health the venous blood of the ciliary body and iris is carried off chiefly by the veins of the choroid, and leaves the interior of the eye through the vortex veins ; a very much smaller quantity emerges through the veins which perforate the sclera in the ciliary region. When the flow through the choroidal veins is embarrassed by an increase of pressure in the chambers, that through the anterior ciliary veins is augmented. A sudden access of high pressure causes intense engorgement of all the external vessels, with more or less chemosis, swelling of the lids, and even proptosis. In less violent attacks the visible injection is confined to the ciliary zone and the larger vessels extending backward from it. In chronic glaucoma there is usually no abnormal redness of the eye beyond some enlargement of the main anterior ciliary vessels, arteries as well as veins. The arteries become hypertrophied in consequence of the increased resistance to the entrance of blood into the eye. They are to be distinguished from the veins by their greater tortuosity, by their very abrupt disappearance at the points where they perforate the sclera, by the greater pressure required to empty them by the finger, and by the re-establishment of the current in a direction from the equator towards the cornea. In chronic glaucoma they are more prominent and more difficult to empty by finger-pressure than in health.

Pain.—Pain is usually proportionate to the vascular disturbance. In acute glaucoma the sensitive ciliary processes are swollen by congestion and at the same time squeezed, and the tunics are put suddenly on the stretch. Severe and sometimes agonizing pain is the result. In chronic glaucoma the rise of pressure is gradual, and the vessels and nerves have time to adapt themselves to the altering conditions. Pain is often entirely absent, or occurs only in the last stage of the disease when attacks of congestion supervene.

Cloudiness of the Cornea.—When the intra-ocular pressure and the tension of the tunics are suddenly raised, the lymph-streams which normally

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traverse the cornea in a radial direction[1] are hindered in their flow, and a condition of œdema is set up. Minute drops of fluid collect beneath the epithelium and between the fibres immediately under Bowman's membrane.[2] This causes a visible cloudiness of the cornea and a peculiar obscuration of vision. (See page 664.) It differs from every form of inflammatory opacity in the rapidity with which it appears and disappears in connection with a rise-and-fall tension. It differs from the opacity which appears when the freshly excised eye of an animal is squeezed between the finger and thumb in the fact that it does not, like this latter, appear and disappear with absolute suddenness and without actual tissue-change, but needs a definite, though short, time for its development and removal. When high pressure is very long continued, the corneal epithelium often thickens and partially separates in vesicles or blebs; the opacity is then denser and more permanent. When, as in simple chronic glaucoma, the increase of tension is very gradual, the œdema of the cornea does not occur.

Anæsthesia of the Cornea.—During acute attacks and in the later degenerative stages there is a partial loss of sensibility in the cornea, due probably to the maceration and compression of the nerve-filaments by the fluid collected in the canals in Bowman's membrane, and to their rupture when the epithelium is raised from its surface.

Dilatation of the Pupil.—The size of the pupil varies to some extent with the amount of blood in the vessels of the iris,—an afflux causing diminution, as after the escape of the aqueous through a corneal puncture; an efflux causing enlargement. The dilatation which, in the absence of synechia, accompanies every rapid rise of pressure probably depends in part on a lowering of the blood-supply to the iris. In typical acute glaucoma it is further promoted by the compression of the iris-base between the turgid ciliary processes and the cornea. This compression affects the nerves as well as the vessels, and the dilatation ultimately becomes permanent through paralysis and atrophy of the sphincter. The oval dilatation seen in many cases probably shows that the compression has been more severe at some parts of the circle than at others. In chronic glaucoma the case is different. The pressure rises gradually, the vessels of the iris have time for compensatory hypertrophy, and the blood-supply is maintained; there is, moreover, little, if any, pinching of the iris-base. The pupil in these cases dilates but little, and sometimes not at all; if the fellow-eye be healthy, it may remain of normal size and consensually active, even after the eye is blind. The loss of retinal sensibility is an auxiliary, though, as the fact last mentioned shows, a less important cause of the dilatation of the pupil.

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[1] Pflüger, Zur Ernährung der Cornea, Klin. Monatsbl. f. Augenheilk., March, 1882; abstract in Ophth. Review, vol. i. p. 246; Gruber, Von Graefe's Archiv, xl., iv., 1894; and abstract in Ophth. Review, vol. xiv. p. 1, 1895.

[2] Fuchs, Ueber glaukomatöse Hornhauttrübung; Von Graefe's Archiv, xxvii., iii., S. 66; abstract in Ophth. Review, vol. i p. 126.

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Loss of Accommodation.—The range of accommodation is usually diminished by the onset of glaucoma, probably because the increase of pressure in the eye puts the choroid more tightly on the stretch and thereby increases the resistance which it offers to the contraction of the ciliary muscle. The pressure which falls on the muscle itself must further impede its action. In the later stages the muscle undergoes atrophy.

Changes in the Depth of the Anterior Chamber.—Shallowing of the anterior chamber is a common symptom in primary and in some nearly allied varieties of glaucoma,—viz., in those associated with tumors of the choroid and with retinal hemorrhage. Deepening of the chamber is characteristic of the secondary glaucoma of serous cyclitis and of congenital buphthalmos. In cases of the former type the lens is pushed forward by retention of fluid in the vitreous chamber; in the latter it is pushed backward by retention in the aqueous chamber. (See p. 635.)

Changes of Refraction.—Increase of pressure may influence the refraction in several ways. Assuming emmetropia to be the initial state, forward displacement of the lens tends to produce myopia, while increased tension of the zonula tends in the opposite direction. Elongation of the globe—a usual result of glaucoma in early life—often induces a high degree of myopia; flattening of the cornea, which sometimes accompanies it, diminishes this effect. In the great majority of cases the refraction, if altered at all, is increased. It is often higher by one or two D. during a glaucomatous attack than before or after it.[1] The astigmatism commonly found after treatment by iridectomy is due, of course, to the operation. The general increase of refraction sometimes found with it is due to a persistent forward displacement of the lens.

Excavation of the Optic Disk.—Under continued excess of pressure the optic papilla is transformed into a cup. The lamina cribrosa,—the sieve-like part of the sclera which gives passage to the optic nerve fibres,—being the weakest spot in the wall of the eye, is displaced backward, together with the nerve-fibres and blood-vessels which it supports. The firmer ring of sclera around it withstands the pressure and sharply limits the area of excavation. The nerve-fibres, being bent and stretched over the unyielding margin, suffer atrophy and loss of bulk, and a deep undermined cup results. (Fig. 29.) Cupping is not to be found during or after a first attack of acute glaucoma, for the atrophy takes time; but even in such cases a depression of the lamina cribrosa has been found in longitudinal sections of the papilla.[2] It can be produced artificially in an excised eye by pressure.[3]

On indirect ophthalmoscopic examination the cupping is made evident by making small lateral movements of the object-lens; the vessels in the

[1] Schoen, Archiv f. Augenheilk., 1893, p. 292.

[2] Brailey, Royal Lond. Ophth. Hosp. Reports, vol. ix. p. 208.

[3] Laker, Klin. Monatsbl. f. Augenheilk., May, 1886, S. 187; and Ophth. Review, 1886, p. 130

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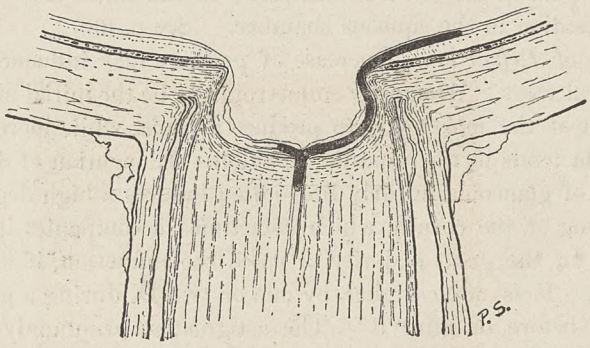
plane of the retina have a greater apparent movement than those at the bottom of the cup, and seem to outrun and travel in front of them. On direct examination it is manifested by the difference of refraction between the margin and the bottom of the cup, and its depth may be roughly estimated from the fact that a difference of three D. corresponds to a difference of level of about one millimetre. The floor of the cup is paler than the normal disk and more distinctly cribriform, through atrophy of the nerve-fibres; at its periphery it appears shaded in contrast with the margin of the cup. The sides are more or less hidden by the overhanging margin, so that the vessels visible on the floor are lost to view as they ascend the side, and reappear changed in number and position as they bend round the

FIG. 29.



Longitudinal section through optic disk from a case of advanced chronic glaucoma.

margin to gain the retina. (Fig. 30.) Around the margin there is usually a narrow zone of lighter color than the adjacent fundus,—a circumscribed choroidal atrophy. The vessels are more or less displaced towards the inner side of the cup. When associated with staphyloma posticum,—*i.e.*, with attenuation of the adjacent sclera,—the glaucoma cup is larger in diameter than usual; it appears oval instead of round, in consequence of its obliquity to the axis of the eye; its sides are less undermined; its vessels attenuated by elongation and unusually free from sharp bends or curves. (Fig. 31.)

The glaucoma cup is to be distinguished from the normal central depression of the healthy disk—the physiological cup, which, when large and undermined, somewhat resembles it—by the fact that it involves the whole area of the disk, which the latter does not. (Fig. 32.) It is to be distinguished from the excavation of simple atrophy by its depth and the interruption of the vessels at its margin; in simple atrophy of the disk there is loss of substance and retraction of the surface quite up to the margin, as in glaucoma, but the excavation remains shallow and is never undermined, for the lamina cribrosa is not displaced. (Fig. 33.) In the early stages, however, these distinctions are not always easy to make, and when simple atrophy attacks a disk in which there is already a large

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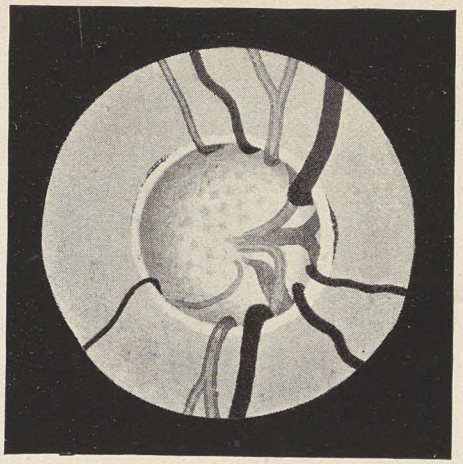
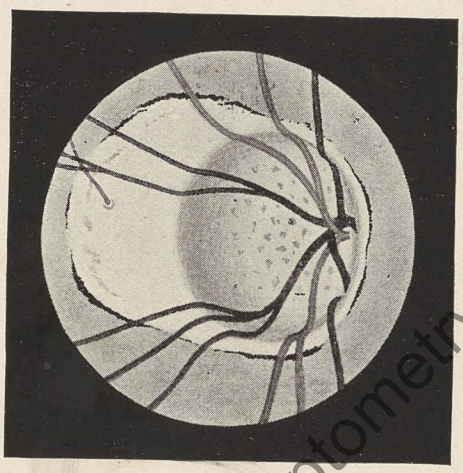
FIG. 30.



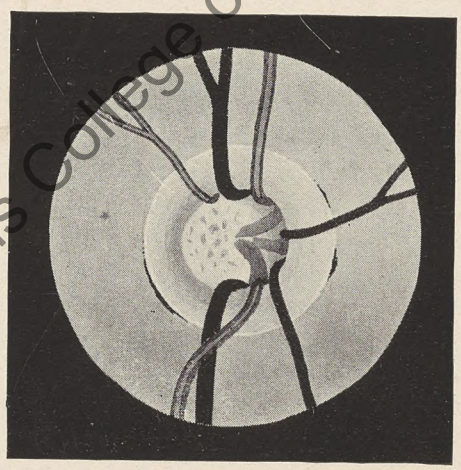
Ophthalmoscopic appearance of optic disk in advanced glaucoma Right eye.

FIG. 31.



Cupped disk of glaucoma associated with posterior staphyloma. Right eye.

FIG. 32.



Physiological cupping of healthy disk. Right eye.

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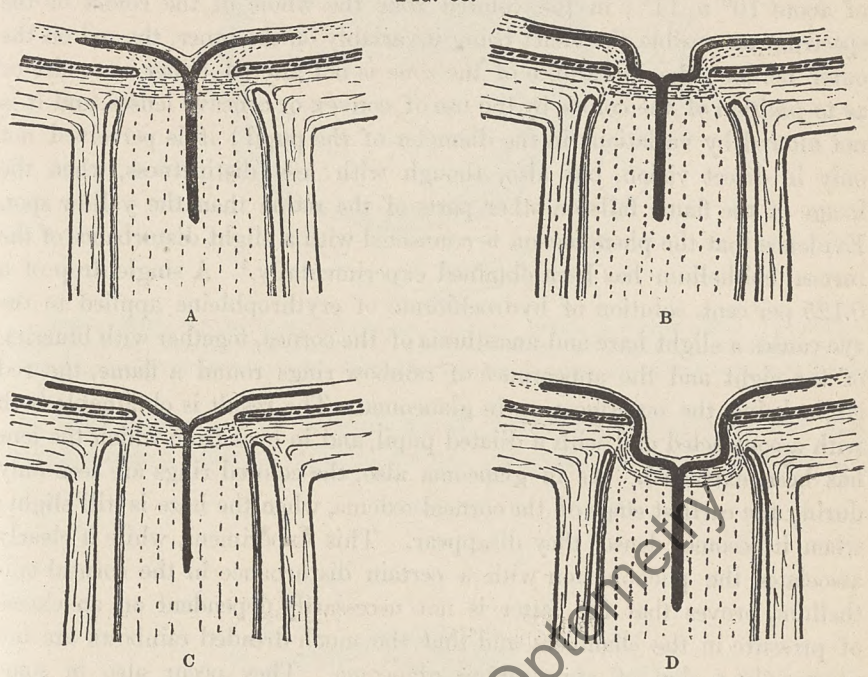
physiological cup, the resulting condition may closely resemble the typical glaucoma cup.[1]

Disturbance of Circulation in the Retina.—The pressure on the retina obstructs both the entrance of the arterial and the exit of the venous stream. The arteries are incompletely filled, the veins congested. Arterial pulsation, invisible in the healthy eye, is often to be seen in the area of the disk, or may be induced by light finger-pressure on the globe; during each diastole of the heart the high vitreous pressure causes arterial regurgitation. The

FIG. 33.



Various forms of depression in the optic disk. (Modified from a diagram by Fuchs.)

A. Ordinary small physiological depression at centre of disk. Lamina cribrosa normal.

B. Unusually large and abrupt physiological cup involving central portion of disk. Lamina cribrosa normal.

C. Shallow saucer-like atrophic depression. Lamina cribrosa normal.

D. Glaucomatous excavation involving whole area of disk. Lamina cribrosa displaced backward.

veins are rhythmically compressed by the transmitted pressure of each incoming arterial wave. This so-called venous pulsation occurs only in a small portion of the vein close to its point of exit, for the blood-pressure is lowest here, and compression at this point prevents the expulsion of blood from the adjacent part of the vein: it is often to be seen in eyes of normal tension, but not so often as in glaucoma. Capillary hemorrhage may result from the obstruction of the retinal circulation. Aneurismal dilatations of the arteries and bead-like varicosities of the veins are occasionally met with.

[1] Schweigger, Archiv f. Augenheilk, xxiii.; and abstract in Ophth. Review, 1891, vol. x. p. 234.

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Impairment of Vision.—The pressure-changes above described are productive of disturbance of vision in several different ways.

The early transient œdema of the cornea causes a transient dimness of sight in the daytime and an appearance of a ring of rainbow colors around every luminous flame at night. The rainbow phenomenon has the following characters:[1] the flame is seen with nearly normal clearness; around it is a dark non-luminous zone, the breadth of which corresponds at all distances with an angle of 4° to 5°; surrounding this is the colored zone, which has a breadth equal to an angle of 2° to 2.5°, and a total diameter of about 10° to 11°; in the colored zone the whole of the colors of the spectrum are visible, the violet being invariably on the inner, the red on the outer border; the appearance of the zone is not altered, either as to size or as to position of the colors, by the use of convex or concave lenses, and it is not altered by variations in the diameter of the pupil; it is perceived not only in direct vision, but also, though with less distinctness, when the image of the flame falls on other parts of the retina than the yellow spot. Evidence that the phenomenon is connected with a slight disturbance of the corneal epithelium has been obtained experimentally.[2] A single drop of a 0.125 per cent. solution of hydrochlorate of erythrophleine applied to the eye causes a slight haze and anæsthesia of the cornea, together with blurring of the sight and the appearance of rainbow rings round a flame, the red circle being the outermost, as in glaucoma. The result is obtainable both with a contracted and with a dilated pupil, and in eyes from which the lens has been extracted. As in glaucoma also, the colored rings are seen only during the earliest stage of the corneal œdema, when the haze is still slight; when it becomes denser they disappear. This experiment, while it clearly associates the phenomenon with a certain disturbance in the corneal epithelium, proves that the latter is not necessarily dependent on an excess of pressure in the chambers, and that the much-dreaded rainbows are not necessarily a sign of approaching glaucoma. They occur also in some forms of conjunctivitis, and perhaps in some other congestive conditions not connected with increased pressure.

The lowering of the retinal circulation which attends a rapid onset of high pressure in the eye impairs the sensibility of the retina. In the healthy eye it is easy, though not, perhaps, very safe, to abolish the whole field of vision by external pressure, and accurate experiment with graduated pressure in subdued light shows that before this result is reached sensibility is lowered over the whole area of the retina, and that it is less easily abolished in the region of the macula than elsewhere.[3] The retinal paralysis which attends the onset of acute glaucoma is of this character. It appears to depend chiefly on the circulatory disturbance in the retina, and perhaps

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[1] Laqueur, Von Graefe's Archiv, xxvi., ii., S. 7.

[2] Treacher Collins, Ophthalmic Review, July, 1890, p. 196.

[3] Author's Jacksonian Essay on Glaucoma, London, Churchill, 1879, p. 88.

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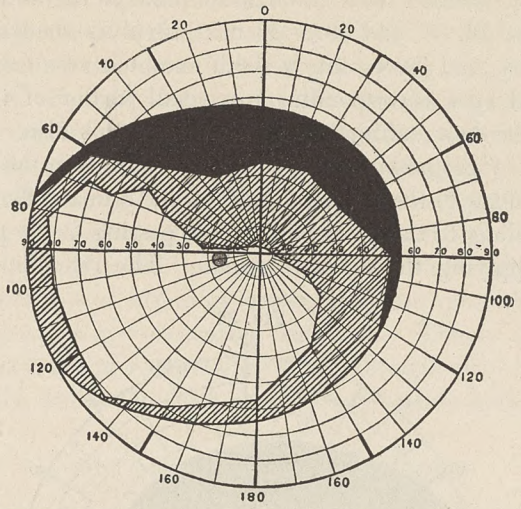
in the choroidal plexus which nourishes the percipient layers; perhaps also to some extent on direct compression of the nervous structures.

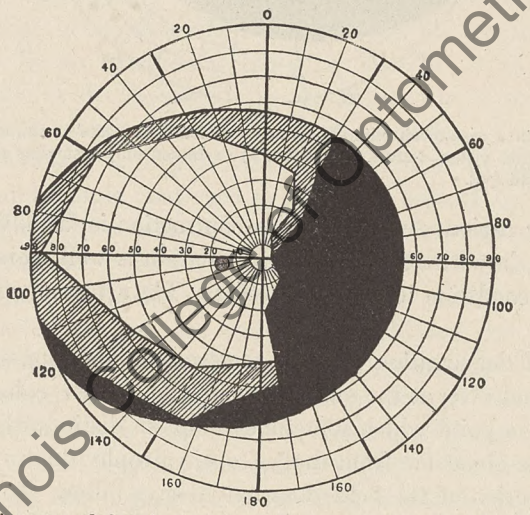
FIG. 34.



Field of vision in a case of chronic glaucoma; left eye. Patient aged seventy-five. The blackened area shows the contraction discovered at the first examination; the shaded area, that which was lost during the following twelve months. During the same period central vision sank from 6-6ths to 6-8ths.

The gradual rise of pressure which characterizes simple chronic glaucoma causes a different kind of retinal paralysis. The centre of the field

FIG. 35.



Field of vision in a case of chronic glaucoma; left eye. Patient aged fifty-seven. The blackened area shows the contraction discovered at the first examination; the shaded area, that which was lost during the following two months. Central vision, 6-18ths.

retains for a time normal or nearly normal vision, while the periphery progressively contracts. The contraction is discoverable at first only in

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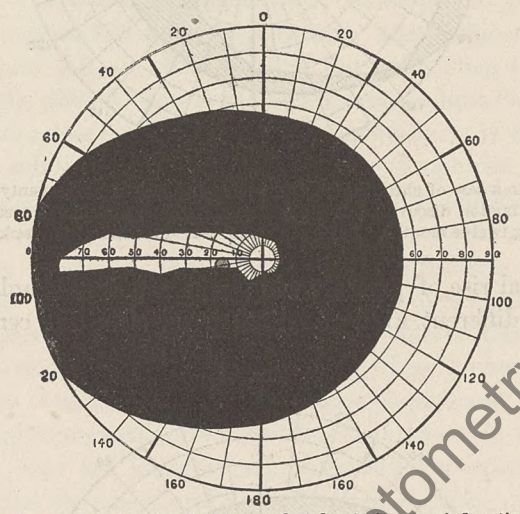
subdued light. In its most typical form it begins at the inner margin of the field of vision, involves the inner upper and lower portions before the outer, and gradually reduces the sentient area to a small oval or slit-like field extending outward from the fixation-point to the blind spot and beyond it. (Figs. 34, 35, and 36.) It next involves the fixation-point and the adjacent area, and leaves only a small excentric area extending outward from the blind spot, corresponding to a small portion of the retina on the nasal side of the disk; ultimately it annihilates this also. It is probably the expression of progressive damage of nerve-fibres in the excavated disk, those which belong to the temporal half of the retina suffering earlier than those which belong to the nasal half; those passing to the periphery earlier than those supplying the macular region. The retention of good vision

FIG. 36.



Field of vision in a case of chronic glaucoma of at least six years' duration; left eye. Patient aged fifty-five. Central vision, 6-12ths. The other eye retained only a smaller excentric field to the outer side of the blind spot.

in the macular region, even when the contraction is far advanced and the pressure high, shows that the retinal circulation is well maintained as compared with its condition in acute glaucoma. The arteries undergo protective hypertrophy.

The line of demarcation between the sentient and non-sentient areas is well defined, and even at the outer limit of the former color-vision is usually retained,—a point which sometimes helps to distinguish the contracted field of chronic glaucoma from that of optic atrophy due to other causes.

The contraction of the field does not always follow the typical course described above. In one hundred cases carefully examined the defects in the field were as follows:[1] defect in nasal portion only, twenty-seven cases; predominating in nasal portion, forty-four cases; loss of whole field,

[1] Bunge, Ueber Gesichtsfeld, etc., Halle, 1884.

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excepting a peripapillary oval, four cases; loss of whole field, including fixation-point, but excepting small temporal area, nine cases; central or paracentral scotoma, with or without slight restriction of nasal periphery, four cases; restriction upward only, two cases; concentric restriction, six cases; preponderance of defect in temporal half of field, four cases. It will be seen that of these one hundred fields only the last sixteen were atypical. When the field is tested by the method of Bjerrum,—*i.e.*, by using a much smaller test-object or a much greater distance than is ordinarily employed,—defects are found which would otherwise escape detection, and have, it is said, the peculiar characteristic that the defective area, wherever situated, is always in direct continuity with the blind spot.[1]

These two types of retinal paralysis are often combined: an acute glaucoma initiates the loss of vision by obstructing the circulation; prolonged pressure completes it by excavating the disk. Timely removal of the pressure will do much to restore what is lost through recent obstruction of the blood-supply, but the loss due to atrophy of nerve-fibres is irreparable.

CLINICAL TYPES OF GLAUCOMA.

Acute primary glaucoma is frequently mistaken by unskilled persons for an ordinary inflammation of the eye, an erysipelas, or a neuralgic attack. The mistake is disastrous, for it leads to irreparable loss of sight. The attack is often, but not always, preceded by one or more transient attacks of dimness and rainbow vision. In some cases it arises without warning of any kind in an eye which, so far as the patient can tell, has previously been free from all disorder. The premonitions are less frequently absent in the younger than in the older class of sufferers. The exciting cause appears frequently to be an exhausting illness, a chill, or a time of exceptional fatigue, anxiety, or sleeplessness; the use of atropine or some other mydriatic to the eye is sometimes immediately responsible. The eye is attacked more or less suddenly with pain: this increases hour by hour, extending to the forehead and temple, and even to the whole side of the head and face. Vomiting sometimes occurs, and in a few hours the patient is utterly miserable and prostrate. Vision is greatly impaired. By the time the surgeon sees the case the changes are already striking and characteristic. The eyelids are somewhat swollen, the conjunctiva reddened and perhaps œdematous; the subconjunctival vessels are much injected, especially the circumcorneal plexus and the main trunks connected with it. In very severe cases a protrusion of the eye is discoverable. The tears flow, and the eye, though perhaps nearly blind, appears intolerant of light. The cornea has lost its polish, and looks like a glass which has been breathed upon. If high tension has already persisted some time, its sensitiveness to touch is diminished. The pupil is more or less dilated and totally inactive,—a condition which at once indicates that the case is not one of ordinary inflammation. The

[1] Bjerrum, Nordisk Ophth. Tidsskrift, and abstract in Ophth. Review, vol. ix. p. 104, 1890.

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anterior chamber is abnormally shallow. The eyeball is decidedly harder than its fellow under the finger-test. This establishes the diagnosis.

The vision of the affected eye is always much impaired; it may in the course of a day or two, or even sooner, be reduced to a bare perception of light near the middle of the field, or it may be totally extinguished. On attempting an ophthalmoscopic examination we are baffled, in spite of the dilatation of the pupil, by the cloudiness of the cornea. If the optic disk can be discerned, the arteries will be seen to be somewhat reduced in size and probably pulsating, the veins engorged, and their main trunks, close to the point of emergence from the disk, collapsing with each arterial pulse. Extravasations of blood are occasionally discoverable in the retina or in the choroid.

In the absence of treatment, the acute symptoms usually last for several weeks and then gradually subside. With their subsidence there may be some slight return of vision, but the eye remains hard and the remnant of sight is gradually abolished.

The intensity of the symptoms in acute glaucoma and the rapidity of their onset vary considerably. While the milder cases form a connecting link with the subacute variety, the most severe well deserve the name "fulminating" applied to them by Von Graefe. The stage of total and irremediable blindness is reached more rapidly in the acute than in the other forms of glaucoma.

Subacute primary glaucoma is characterized by its intermittency. The first few attacks may amount to nothing more than transient obscuration with rainbow vision. Gradually the recurrences become more frequent and severe and the remissions less complete, and by degrees a persistent congestive glaucomatous condition is established. The tension remains in excess; excavation of the disk and contraction of the field begin. The further course of the disease is still marked by exacerbations of pain and injection, and ultimately it leads, less rapidly than acute glaucoma, but not less surely, to total blindness.

The term inflammatory, commonly applied by Continental writers to the congestive forms of glaucoma described above, is here intentionally omitted, as conveying a false impression as to the essential nature of the morbid process.

Chronic primary glaucoma (glaucoma simplex) begins almost imperceptibly, progresses slowly, with little tendency to exacerbation or remission, and leads, in the course of months or years, unless arrested by treatment, to total blindness. The patient is usually at least fifty years of age. He complains of failing sight in one or both eyes. One eye is usually affected before the other, and with unobservant persons it sometimes happens that the eye first affected becomes blind, or nearly so, before its owner discovers that anything is going wrong. In a large majority of cases the second eye is affected sooner or later in like manner. We sometimes obtain a history of periodic obscurations and rainbow vision; more frequently such history

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is wanting, and no exact date can be assigned to the onset of the disorder. A history of nervous exhaustion from one cause or another is frequently forthcoming.

Externally the eyes exhibit little or nothing amiss, except perhaps a slight enlargement of the chief anterior ciliary vessels. The anterior chamber is often shallow, but not more so than in many healthy eyes at the same time of life. One or both pupils may be somewhat dilated and sluggish, but this condition is by no means constant. An eye already blinded by typical chronic glaucoma may present a non-dilated and consensually active pupil. The lens, on simple inspection, may appear wanting in transparency when it is not really so. The inexperienced observer may well be astonished to find that in a glaucomatous eye he can sometimes see every detail of the fundus with the ophthalmoscope through a crystalline lens which to the naked eye looks like a cataract almost ready for extraction. Unfortunate mistakes of diagnosis sometimes arise from this cause.

The tension of the eyeball is increased. The excess is at first very slight, and may be discoverable only on repeated examination at different times of day. In such cases the tonometer is helpful. In the later stages it may amount to almost stony hardness, although, by reason of its slow advance, there is no pain and little external sign of disease. The optic disk is cupped; it presents the characteristic appearances of excavation with displacement of the vessels which have been described. (Page 661.)

On examining the functions of the eye we find the range of the accommodation relatively small, if not totally wanting; the refraction, in the majority of cases, hypermetropic; the acuteness of vision, as tested by Snellen's letters, more or less impaired, though occasionally, even when the disease is far advanced, the macula retains nearly normal acuteness; and the field of vision typically contracted. (Page 666.)

The diagnosis of chronic glaucoma depends chiefly on the excess of tension, the excavation of the disk, and the contraction of the field, and in cases of doubtful tension on the two last named. In the absence of decided increase of tension, chronic glaucoma is apt to be mistaken for simple atrophy of the optic nerve, and the differential diagnosis is sometimes very difficult. The distinguishing characters of simple atrophy are the shallowness of the cup, the relatively greater loss of color-vision, and the less regular distribution of the blind area in relation to the optic disk. A definite history of rainbow vision, when obtainable, favors the diagnosis of glaucoma.

Absolute Glaucoma and Glaucomatous Degeneration.—When the stage of blindness is reached, glaucoma is spoken of as absolute. In this stage the ultimate effects of high pressure in the eye are witnessed. The symptoms vary according to the previous character of the disease. If it have been acute or subacute, the anterior ciliary vessels are large, tortuous, and of deep purplish color, and stand out with especial prominence by reason of the atrophy of the conjunctiva. The cornea is hazy, its epithelium thick-

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ened, rough, and sometimes raised in blebs, its sensibility lowered or abolished. If the iris is visible, it is seen to be reduced to a narrow circle by reason of wide dilatation of the pupil, its characteristic markings are gone, and its periphery is sometimes visibly in contact with the cornea, the surface being altered in color by destruction of the epithelium. Large tortuous vessels are sometimes to be seen in the iris. The lens, which at an earlier stage often appears more or less opaque when it is not really so, becomes truly cataractous, the opacity ultimately attaining the dense white or yellowish character which in all forms of secondary cataract denotes advanced disease of the ciliary body; it is then unpleasantly conspicuous by reason of its advanced position and the largeness of the pupil. Pain is apt to continue long after sight is lost, and in this stage it is usually for the relief of pain that advice is sought. Subjective visual sensations are not infrequent long after the eye is blind, and occasionally lead the patient to the surgeon in the vain hope that they indicate a chance of improvement.

If the course of the glaucoma have been chronic throughout, without pain and congestion, the external appearance of the eye may be little altered, even in the absolute stage. The symptoms are those of chronic glaucoma, already described. The diagnosis is made at once from the hardness of the eye and the excavation of the disk. Sooner or later, however, further changes, including pain and congestion, usually ensue.

The last stage is marked by changes in the size and shape of the eyeball. The tunics become gradually thinned and extended; the curvature of the cornea flattens and approximates to that of the sclera; the conjunctiva atrophies. Instead of a general enlargement of the globe, or simultaneously with it, there is often a more circumscribed distention of the sclera at one or more places, usually the ciliary region, the equator, or the posterior pole. This thinning of the tunics is apt to lead to rupture during any muscular effort, such as coughing or sneezing, which is accompanied by forcible contraction of the eyelids. The rupture is followed by profuse hemorrhage, and the eyeball ultimately collapses. If, on the other hand, the disorganization of the ciliary body have proceeded in advance of the thinning of the sclera, changes of an opposite kind occur,—loss of tension and shrinkage. The cornea contracts in all diameters, the sclera is indented by the traction of the tendons, and a small, atrophic, shrunken eye remains. Even in this condition there is sometimes a liability to attacks of pain. Ulceration and rupture of the cornea, extrusion of necrosed tissue, and even hemorrhage of considerable amount may complete the destructive process.

Hemorrhagic glaucoma belongs properly to the secondary group, but much resembles the congestive primary forms, and is sometimes indistinguishable from them. Cases in which the onset of glaucoma has been preceded by hemorrhage within the eye have a special importance from the clinical point of view, for the operative treatment appropriate to primary

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glaucoma is apt, in them, to lead to further hemorrhage and consequent failure.

Secondary glaucoma, whatever be the pre-existing disease, is a formidable complication of it; in certain disorders of the eye it is one for which the surgeon should be always on the watch, as its occurrence may at any time necessitate a prompt alteration of treatment. The essential and characteristic symptom is the increase of tension. Vascular injection, pain, and impairment of vision, if already present, are aggravated by the access of high pressure in the eye. The pupil, if it be free, dilates. The field of vision, if not already lost, contracts. The optic disk, if visible, will be seen in course of time to suffer excavation. In short, we may say that the symptoms of secondary glaucoma are those of the primary disease more or less altered or concealed by various morbid changes in the eye.

TREATMENT.

The progress of glaucoma can be arrested only by measures which lower the tension of the eye, and these must be employed in the earlier stages if permanent loss of sight is to be avoided. In the absolute stage treatment can avail nothing beyond relief of pain, and this end is attainable more certainly, speedily, and safely by complete removal of the useless organ than in any other way. The treatment of various forms of secondary glaucoma has been incidentally referred to already in a previous section. The following pages deal mainly, though not exclusively, with the treatment of the primary disease.

Glaucoma usually calls for operative treatment, for the due escape of the intra-ocular fluid can seldom be permanently re-established by any other means; but there are some cases which can be successfully treated without operation, and there are very many in which certain auxiliary measures are of great value.

PALLIATIVE TREATMENT.

Eserine (*physostigmine*) sometimes rapidly relieves the high tension;[1] it is the antagonist of atropine, which, as already stated, sometimes induces or aggravates high tension. Its use should be guided by a clear conception of its mode of action. It is noteworthy that neither myotics nor mydriatics cause any decided change of tension in the normal eye, and that such small changes as they do produce are the opposites of those here in question,—eserine tending to raise and atropine to lower the intra-ocular pressure. Their action in relation to glaucoma depends on the abnormality in the position of the iris. Eserine, by contracting the sphincter of the pupil, thins the iris, flattens its folds, and pulls upon its peripheral insertion. If the filtration-angle is compressed, it tends to reopen it. Accordingly, it is

[1] Adolph Weber and Laqueur appear to have discovered the curative action of eserine almost simultaneously and independently of each other. See Von Graefe's Archiv, xxii., iv., S. 216.

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chiefly useful when this compression is recent and slight. In the sudden but comparatively mild attacks which come and go during the premonitory stage of primary glaucoma eserine acts with admirable effect. In severe acute attacks also it may be useful if applied without delay; but in such cases the filtration-angle is very firmly closed and the sphincter of the pupil is soon paralyzed; hence the stage during which it can relieve is soon passed. In chronic non-congestive glaucoma it often lowers the tension for a time, but the improvement is seldom great or lasting.

In some forms of secondary glaucoma also—for example, in that which follows retinal hemorrhage and that which is due to lateral displacement of the lens—eserine sometimes acts just as in primary glaucoma.

When eserine is unable to contract the pupil, and thereby to increase the patency of the filtration-angle, it is commonly quite useless, and when useless it is likely to be harmful, for it increases the hyperæmia and often causes pain. It should therefore be used in the minimum amount and with the minimum frequency which suffice to contract the pupil and to keep it contracted. A two-tenths per cent. solution of sulphate of eserine in distilled water (about one grain to one ounce) is probably the strongest which need ever be used, and a much weaker solution is often better. A one or two per cent. solution of nitrate or hydrochlorate of pilocarpine is preferred by some surgeons to eserine on account of its comparatively feeble action, but it appears to have no distinct advantage over a sufficiently weak eserine solution.

Atropine is harmful precisely in those conditions in which eserine is useful; it induces high tension by dilating the pupil, slackening and throwing the iris into folds, and thereby helping to obstruct the filtration-angle. Hence it is mischievous in the earlier stages of primary glaucoma, and may accelerate the onset in cases of intra-ocular tumor and hemorrhage. When it cannot dilate the pupil, atropine never, probably, raises the tension. In certain cases of secondary glaucoma in which the high tension is due to serous exudation into the aqueous chamber and not to compression of the filtration-angle,—*e. g.*, in serous cyclitis,—atropine, by lessening the inflammation, tends to restore normal tension. The choice between a myotic and a mydriatic is not always easy to make: the one or the other must be employed tentatively, and the eye must be re-examined after an hour or two, or at latest on the following day.

Cocaine, like every other dilator of the pupil, has been known, under predisposing conditions, to induce glaucoma. On the other hand, this drug has the power—invaluable in glaucoma—of contracting the ciliary blood-vessels and diminishing the sensibility of the ciliary nerves,—effects which tend to lower the intra-ocular pressure. By combining cocaine with eserine in such proportions that the eserine shall retain the mastery over the pupil, we get the advantages of both without the disadvantages. A solution containing two-tenths per cent. of sulphate of eserine and one per cent. of hydrochlorate of cocaine (one grain and five grains respectively to

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one ounce of distilled water) is quite safe in this respect, and it is often better to make the eserine still weaker and the cocaine stronger.

Morphine, given subcutaneously or by the mouth, in small doses, will sometimes cut short and will almost always help to alleviate a glaucomatous attack. It eases pain, lowers blood-pressure, lessens secretion, and promotes contraction of the pupil and sleep.

Sleep, even though of very short duration, often dispels the mild premonitory attacks with which primary glaucoma begins. During sleep the pressure in the cerebral vessels falls and the pupil contracts.

Warmth, *food*, and *rest* relieve, just as cold, hunger, and fatigue induce, these early and slight attacks.

Aperients sometimes produce a decided effect on the tension and congestion of the eye in those cases—not very uncommon in women—in which an attack of subacute glaucoma is associated with prolonged constipation.

Ice, applied to the closed lids in the form of iced compresses, or more conveniently enclosed in a thin rubber balloon, will occasionally prove of service in conjunction with other palliative measures.[1]

A judicious combination of these palliative measures, especially the use of eserine and cocaine to the eye and morphine internally, will sometimes rapidly subdue a recent congestive glaucoma, even of severe type, and may for a time restore the eye to an apparently healthy state. The cure, however, will rarely prove permanent, and with each recurrence the treatment is likely to prove less effectual. Palliative treatment is useful chiefly as a means of lessening the severity of the symptoms, of gaining time, and of bringing the eye into a condition more favorable for operative treatment. The immediate benefit, however great, must not be allowed to obscure the fact that in the great majority of cases a lasting cure is obtainable only by timely operation, usually iridectomy.

OPERATIVE TREATMENT.

The urgency for operation varies with the acuteness of the disease. In acute glaucoma, unless decided improvement is gained in a few hours by the measures already mentioned, iridectomy should be performed without delay. A very intense attack may in the course of a few hours reduce vision to a bare perception of light; a prompt iridectomy may restore it nearly to its normal condition; but just in proportion to the brilliancy of such a result is the danger of delay. In the course of a few days the case may become incurable. So long as perception of light remains, and if it have not been absent more than a few days, iridectomy should on no account be withheld. Prostration of the patient is no reason for delay: iridectomy is the surest means of procuring ease and sleep. Thus, a patient of my own appeared, when I first saw her, to be past recovery. The glaucoma, which had begun violently and suddenly, was of fourteen days' duration in one eye, of seven

[1] Lloyd Owen, Middlemore Lectures, Birmingham, Cornish Bros., 1890, p. 46.

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days' duration in the other. Neither eye could perceive the light of a candle close to the face in a dark room. An immediate iridectomy in each eye restored a part of the outer half of the field, but not the fixation-point, in the one; nearly the whole of the outer half, together with the fixation-point and central vision of 6-18ths, in the other.

In subacute glaucoma also early operation is important, for each recurrence diminishes the chance of complete restoration. If the visual field, examined during a quiescent interval, be much contracted, complete restoration must not be expected; but great impairment of vision during an attack does not preclude the recovery of good vision after iridectomy.

In chronic glaucoma operation confers less certain and less obvious benefit. Positive improvement of vision is seldom to be hoped for; the most that iridectomy can do is to prevent further loss. Even this is not obtainable in every case. The operation sometimes fails to arrest the course of the disease, and occasionally induces a painful aggravation with rapid loss of whatever sight remains. It is the only means by which the eye can be saved from certain blindness, but it is not a certain means, and this should be fully and carefully explained to the patient, or in some cases preferably to his friends, before it is undertaken. If both eyes are affected but both still retain some sight, the worse should be operated on first. The result may decide the treatment of the other. The prospect of success is greater in the earlier than in the later stages, but there is never a sudden urgency for operation. Even in advanced cases it is sometimes wise to re-examine once or more at intervals of a few weeks, so as to make certain that the disease is progressive before proceeding to operate. It is well to remember, however, that such patients, when lost sight of, are apt to neglect the progress of their malady until help is no longer possible.

There is some difference of opinion as to the propriety of operating in simple chronic glaucoma. Some surgeons regard the uncertainties as so grave that they advise their patients rather to accept the gradual approach of certain blindness. This attitude cannot, I think, be justified. Cases of definite arrest of chronic glaucoma, with retention of good vision over many years, are known probably to all experienced operators. Cases of definite reduction of tension, with retention of good vision lasting so long as the patient remains under observation, are quite common. Cases in which the loss of vision is definitely hastened by the operation are comparatively rare. Nettleship, reviewing his experience over a long period, concludes that it is a clear duty to operate, even though the malady be far advanced, unless the patient's state of health definitely contra-indicates.[1] My own experience, especially since I have adopted the addition of a scleral puncture (see page 680), is in favor of operating so long as the eye retains any sight worth saving. The benefit conferred by operation in cases of chronic glaucoma must be estimated from the resulting condition of the

[1] Royal Lond. Ophth. Hosp. Reports, vol. xii. p. 97.

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tension and the field of vision rather than from slight changes in acuteness. The enlargement of the pupil and the altered curvature of the cornea produced by an iridectomy frequently involve some lowering of acuteness, even after careful correction of the astigmatism, but this is of small moment when compared with the arrest of the disease, as indicated by a reduced tension and a non-contracting field. The permanent benefit will be revealed rather by the tonometer and the perimeter than by the test-types.

Mode of performing Iridectomy. — The operation consists in making an incision which opens the anterior chamber near its periphery and removing as completely as possible the corresponding segment of the iris. In congestive cases a general anæsthetic is usually required; in simple chronic glaucoma cocaine will usually suffice. As in many other ophthalmic operations, the position of the eye may be advantageously controlled by letting the patient look at a lighted candle held in the requisite position. The incision is made with a small linear cataract knife or with a broad keratome. In my own opinion, the linear knife is preferable, in that it enables one to make a more peripheral incision than can be safely made with the lance knife, and to modify the length and the position of the incision, according to the space available after the point has appeared in the anterior chamber, by making the counter-puncture a little farther forward or backward, as may be found practicable. The incision is usually placed at the upper part of the cornea. The puncture is in the sclera, at least one millimetre from the corneal margin; the counter-puncture is at a corresponding point; the distance between puncture and counter-puncture externally in a straight line is from seven to nine millimetres, according to the size of the cornea and the depth of the anterior chamber. When the iris lies very close to the cornea it is impossible to make a full-sized incision. In passing across the chamber the knife is kept parallel with the plane of the iris; in cutting out the edge is turned somewhat forward, but must come out well in the sclera and raise a conjunctival flap. The iris is seized with the small forceps, drawn out through the incision, and lightly pulled to the right and to the left, so that its base may be disengaged from the cornea. It is then divided with scissors close to one end of the incision, drawn towards the other end, and, if possible, torn at its root; is again drawn away from the angle of the wound so that it may not be pinched and incarcerated; and is removed with a second snip of the scissors. Instead of making the incision upward, some operators place it at the part of the circle where the iris appears to respond most readily to eserine, on the ground that complete removal of the iris-segment is more likely to be attained there than elsewhere. The upward incision has the advantage over incisions which are placed laterally that it can be made with the linear knife, and that the resulting coloboma lies under the upper lid.

For reasons which will be explained later, I have for several years adopted the plan of slackening the eye by a scleral puncture immediately before making the iridectomy.

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Mode of Action of Iridectomy.—It is interesting to note that Von Graefe, to whose clinical acumen we owe this most beneficent remedy, declared himself, in his last utterance on the subject, unable to explain its action;[1] yet the rules which he laid down for its employment are completely confirmed by the advanced pathology of the present day. We now know that iridectomy arrests the progress of glaucoma either by causing the normal filtration-outlet of the eye to reopen or by establishing a more or less abnormal one in its stead.

In recent congestive glaucoma iridectomy appears to act as follows. The escape of the aqueous humor and the simultaneous advance of the lens immediately slacken the whole eyeball. Fluid drains away for some hours at least, and the overfilled vitreous chamber is depleted. The obstructed circulation in the uveal tract is relieved by the removal of the pressure, and in many cases is further relieved by a free escape of blood from the divided vessels of the iris. The turgid ciliary processes recede and cease to compress the base of the iris, and with the re-establishment of the anterior chamber the filtration-angle reopens. (Fig. 37.) The normal outlet resumes its

FIG. 37.

From an eye permanently cured of acute glaucoma by iridectomy. (After a drawing by Fuchs.)—The patient was a woman, aged sixty-six. Both eyes were attacked by acute glaucoma at intervals of one year, and both were permanently cured by iridectomy. They were examined after death, seven years later. In both eyes the attack had been of short duration (in the second eye only two days), so that, except for the coloboma, it had probably altered the conditions of the eye but little. The ciliary body was large, both as regards the muscle and the processes. The processes extended almost to the lens on the one side and to the iris on the other. The angle of the anterior chamber was narrowed by the altered position of the iris-base, so that a slight swelling of the processes would have pressed the iris against the cornea. The circumlental space was remarkably small, not through enlargement of the lens, but through enlargement of the ciliary processes. The tissues of the ciliary body were normal, not inflamed. (Von Graefe's Archiv, vol. xxx., iii., S. 128.) See also Treacher Collins on a case of recent acute glaucoma cured by iridectomy. (Hunterian Lectures, Lancet, December, 1894.)

function, and, as regards the future, the absence of the iris-segment is to a certain extent a safeguard against the recurrence of a similar blockade.

In congestive glaucoma of longer standing restitution of the filtration-angle is often unattainable, but even in such cases a well-made iridectomy will sometimes free the ligamentum pectinatum in the region of the wound by tearing away the iris from its root. In many of these cases, however,

[1] Von Graefe's Archiv, vol. xv., iii., S. 252.

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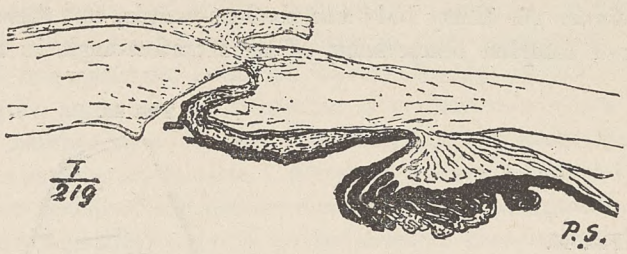
and in chronic glaucoma also, the success of an iridectomy depends on the formation of an artificial filtration-channel,—a minute, permanent, corneo-scleral fistula. (Figs. 38, 39, 40, 41.) The lips of the wound, especially

FIG. 38.



From an eye which was cured of subacute glaucoma by iridectomy and retained useful vision for fifteen years.—Thin section near extremity of cicatrix,—left extremity as seen in Fig. 39. Lips of wound remain ununited, and a tag of iris lines the aperture. At other parts of the incision there is a solid cicatrix.

the inner lips, do not unite completely, but remain more or less separated by the prolapse between them of a fold or tag of iris. The aqueous continues to escape at this point into the subconjunctival tissue, and is thence absorbed. One or more fistulæ lined by iris-tissue are thus permanently established. The overlying conjunctiva presents an œdematous or pearly appearance, and is more or less elevated by the collection of fluid beneath it. Finger-pressure carefully applied to the eye day after day during the healing process[1] seems in many cases to aid in keeping the tension low, and probably promotes the formation of such fistulæ. Even weeks after an iridectomy, firm pressure with the finger will sometimes cause an immediate visible extrusion of fluid beneath the conjunctiva, with slackening of the globe, and a case is related in which the patient was accustomed himself to relieve the slight recurrences of glaucomatous symptoms to which he remained liable in this way.[2] In chronic glaucoma, and in the later stages of the congestive forms, a slightly fistulous scar affords the best, if not the only, guarantee against a speedy return of the glaucoma, and is, therefore, a result to be desired. It is noteworthy, however, that fistulous and cystoid cicatrices are not entirely without danger to the eye, for they occasionally afford a starting-point for septic inflammation. When we have learned how to establish with certainty a safe and persistent drainage of the eye, the treatment of glaucoma will be more uniformly successful than at present.

FIG. 39.

Diagram showing the position of the sections. The globe was bisected in the vertical meridian through the middle point of the cicatrix.

[1] Recommended by Dianoux as a supplement to sclerotomy, Archives d'Ophtal., 1883, p. 404.

[2] De Wecker, Thérapeutique Oculaire, Part I., p. 381.

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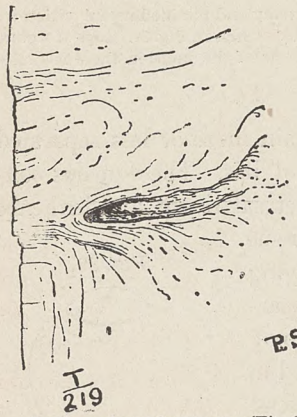
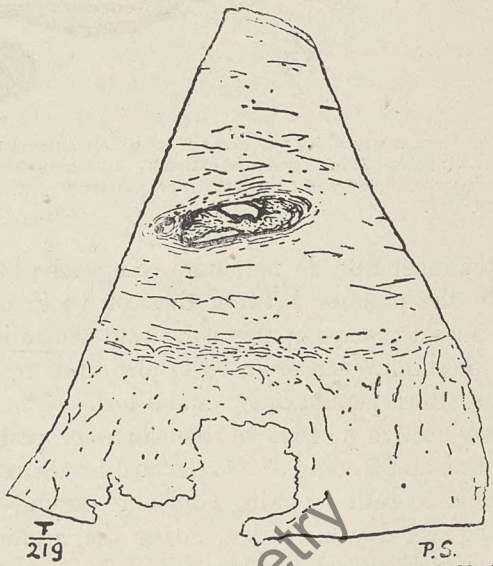
An iridectomy for glaucoma usually leaves a considerable flattening of the cornea in the meridian which is at right angles with the cicatrix, thus producing an astigmatism readily observable with the ophthalmometer or the shadow-test. In other words, there remains a certain degree of ectasia in the region of the cicatrix, and this, according to Snellen,[1] increases the distance between the ciliary body and the lens-margin, and thereby lessens the danger of complete compression of the filtration-angle in the future.

FIG. 40.



Surface view of same eye (Fig. 39), showing subconjunctival channel which was revealed when the prominent conjunctiva covering the right half of the cicatrix was partly snipped off.

FIG. 41.



Section parallel with surface, including right half of cicatrix. The lips of the wound are not united here, but remain separated by iris-tissue and by open channels which lead directly into the subconjunctival space seen in Fig. 40.

By looking obliquely through the coloboma with the ophthalmoscope, one can often distinguish the free margin of the lens and a narrow free space beyond it, and upon the lens-margin one can sometimes see signs of its previous contact with the processes.

Failure of Iridectomy.—Among the causes of failure, profuse hemorrhage within the eye, occurring during the operation or within the following few days, is perhaps the one over which we have least control. It is greatly to be feared where the glaucoma is known to have followed a retinal or other intra-ocular hemorrhage, even of small amount; it may happen in spite of the greatest care where vascular degeneration is extreme; it may arise in any case through violent coughing, sneezing, or other effort tending to congest the eye; it may depend on injury of the ciliary processes by a too deeply placed incision. Its prevention, as far as possible, depends on

[1] Ophthalmic Review, February, 1891, p. 48.

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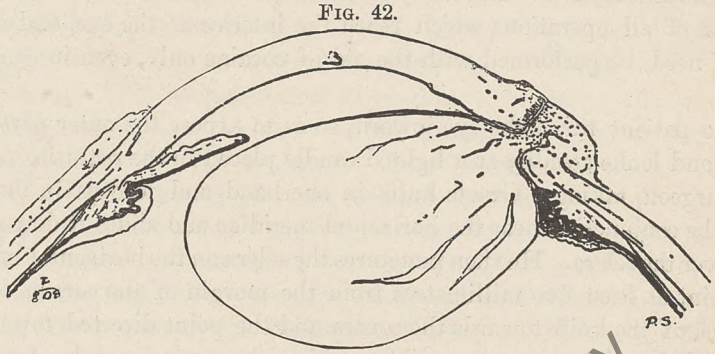
careful regulation beforehand of the patient's condition, with especial regard to the action of bowel and kidneys, sleep, and absence of cough; on gentleness in operating; and on the attainment of rest and tranquillity during the following few days. The most formidable form of hemorrhage is the retro-choroidal; it causes extrusion of the lens and vitreous through the incision, and calls for prompt removal of the eye. Fortunately, it is very rare. Failures due to hemorrhage are quite exceptional.

More frequently the operation fails by effecting neither a reopening of the filtration-angle nor the formation of a vicarious channel. When the anterior chamber is very shallow, it is difficult and sometimes hardly possible to make a satisfactory incision. A corneal incision is commonly ineffectual: it does not permit of satisfactory removal of the iris-segment, and it closes too quickly and firmly, giving an inextensible cicatrix, which affords no

FIG. 42.



From a case of unsuccessful iridectomy.—The operation was performed in a very advanced stage of chronic glaucoma, the eye being nearly blind. The lens-capsule was probably ruptured by pressure against the back of the Graefe knife, though there was no evidence of the mishap at the time.

drainage and no ectasia; high tension soon returns, and no advantage is gained. Moreover, a very shallow chamber with high tension involves danger to the lens. As the aqueous escapes the lens advances, through pressure from behind, and may be ruptured against the back of the knife through the intervening iris. This sometimes happens without the knowledge of the operator, and leads to unexplained loss of the eye. (Fig. 42.) Again, under similar conditions, and without injury of the lens itself, the suspensory ligament may give way and the wound become blocked by the displaced lens. The lens may even be extruded from the eye during the following twenty-four hours.

Of special importance are the cases of so-called "malignant glaucoma" in which, after an apparently perfect operation, the lens advances so as to block the wound. The iris remains in contact with the cornea; there is no escape of fluid from the eye; high tension returns, with pain and conjunctival œdema; and whatever sight remains is quickly lost. This complication may occur in spite of the utmost care in operating; it is probably associated, in some cases at least, with slackness of the suspensory ligament. The eye can be saved only by replacement of the lens in its normal

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position and re-establishment of the anterior chamber by Weber's method. (Page 681.)

Apart from unavoidable hemorrhage, it is evident that the difficulties and dangers which beset the operation depend largely on the high pressure in the vitreous chamber and the advanced position of the lens. Hence the value of a scleral puncture before the knife enters the anterior chamber.[1]

Scleral Puncture: Posterior Sclerotomy.—This proceeding is useful chiefly as an adjunct to iridectomy or sclerotomy. Used alone, it rarely, if ever, effects a permanent cure, but it may with advantage be employed as a preparatory or test operation in certain cases; for example, where there is a known tendency to hemorrhage; in painful glaucoma, where a general anæsthetic is inadmissible; in very advanced cases, where the possibility of recovering useful sight is doubtful. The result may justify and facilitate the performance of an iridectomy a few days later. It is the easiest and quickest of all operations which reach the interior of the eye, and may, in case of need, be performed with the aid of cocaine only, even in congestive cases.

The patient turns the eye inward, so as to expose the outer part of the sclera, and looks steadily at a lighted candle placed in the requisite position. The surgeon, taking a Graefe knife in one hand and forceps in the other, seizes the conjunctiva near the horizontal meridian and slides it downward a little over the sclera. He then punctures the sclera on the horizontal meridian at a point at least five millimetres from the margin of the cornea, keeping the back of the knife towards the cornea and the point directed towards the centre of the globe, so as to avoid touching the posterior pole of the lens. After entering about ten millimetres the knife is slowly withdrawn and at the same time slightly rotated on its axis, so as to give a gaping wound, through which fluid or consistent vitreous escapes. The conjunctiva is then allowed to slide back into its place. The escaping fluid is sometimes watery, sometimes as thick as glycerin, and usually colorless. Yellow fluid probably indicates previous hemorrhage. External bleeding from the puncture, when it occurs, is trivial. Internal bleeding I have discovered only twice in more than sixty cases. Septic infiltration of the vitreous through the puncture has been known to occur, but I have not met with it. Absolute asepsis, and the sliding of the conjunctiva, are the safeguards.

A scleral puncture made immediately before a glaucoma iridectomy—*i.e.*, at the same sitting—appears to me to facilitate the making of a good incision, to diminish the immediate risks which attend it, and to obviate the forcible displacement of the lens forward which sometimes annihilates the effect of a good operation. I have performed this combined operation in more than fifty cases, including many of chronic type in a very advanced stage, and am under the impression that the results have been decidedly

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[1] Author's Lectures, p. 161; and Trans. Internat. Ophth. Congress, Edinburgh, 1894, p. 38.

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better than could have been obtained by iridectomy alone.[1] If not employed as a preliminary step, a scleral puncture should be made whenever the tension of the eye immediately after an iridectomy is found to be insufficiently reduced. When malignant glaucoma is once established, the puncture will not suffice by itself to save the eye. Weber's method must then be adopted.[2] The puncture must be immediately followed by steady pressure on the centre of the cornea by means of a curette or other smooth instrument, continued until the anterior chamber is re-established by the entrance of fluid between iris and cornea. For this more critical manœuvre a general anæsthetic is usually necessary. If Weber's method fail, or if the lens be injured as well as displaced, extraction of the lens affords the only chance of saving the eye. The posterior capsule should then be divided, so as to establish free communication with the vitreous chamber.

Good results obtained by removal of the lens, combined with rupture of the hyaloid membrane, in cases of very advanced glaucoma, and after the failure of iridectomy and sclerotomy, have been recorded.[3]

Sclerotomy.—This operation aims at opening the anterior chamber near its periphery by an incision more or less resembling the first act of an iridectomy, and leaving the iris intact. It is based on the conviction that excision of the iris-segment plays no essential part in lowering the tension of the eye. Experience has shown that every form of primary glaucoma which is amenable to iridectomy will in some instances yield to sclerotomy, but the majority of surgeons are agreed that sclerotomy is less to be trusted, for the reason that during the healing process the iris is apt to occlude the wound, and that even when this does not occur the filtration-angle remains more prone to occlusion in the future than after the removal of the iris-segment. The chief use of sclerotomy is as a supplement to iridectomy when the latter fails to permanently reduce the tension. In the absence of the iris-segment it is a simple operation, and seems preferable in every way to a second iridectomy at the opposite side of the circle. In case of need it may be repeated several times, and, though at first followed by relapse, may in the end give a permanently good result. By some surgeons sclerotomy is preferred as the primary operation in simple chronic glaucoma; but there is no evidence to show that it affords better results. Nettleship, after an extensive trial of sclerotomy in this disease, declares his preference for iridectomy.[4] Snellen, on the other hand, adheres to sclerotomy executed in the special way described below. For glaucoma with deep anterior chamber, as in serous cyclitis and congenital buphthalmos, sclerotomy or paracentesis of the cornea is preferable to iridectomy.

[1] For independent testimony in favor of this proceeding, see Gifford, Opththal. Review, vol. xii. p. 248, 1893; Nicati, Archives d'Ophtal., vol. xi. p. 168, 1891; and several speakers at the Edinburgh Congress, see Transactions, p. 38.

[2] Von Graefe's Archiv, xxiii., i., S. 86.

[3] Rheindorf, Klin. Monatsbl. f. Augenheilk., 1887, S. 148.

[4] Royal Lond. Ophth. Hosp. Reports, vol. xii. p. 100.

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In such cases the incision must be of small extent and repeated several times in case of need, each time at a different part of the cornea. For secondary glaucoma after cataract extraction, sclerotomy combined with division of any membrane which is stretched across the pupil is usually the best remedy. (See page 639.)

Sclerotomy, according to De Wecker's method, is performed as follows.[1] The pupil is fully contracted by eserine or pilocarpine. The incision is made with the ordinary linear cataract knife, and resembles a scleral incision for the extraction of cataract, except that the middle third is left uncompleted. Puncture and counter-puncture lie at one millimetre from the corneal margin, and are so placed that the knife-edge forms a tangent to the extreme margin of the cornea or lies rather deeper than this within the angle of the chamber. After slow evacuation of the aqueous the knife is slowly and steadily withdrawn, leaving more or less of the sclera undivided. Some operators divide the sclera completely and leave only a bridge of conjunctiva. Snellen performs sclerotomy with a broad, bent, lance-shaped knife, according to the method originally proposed by Quaglino, but with a special conjunctival flap. The conjunctiva is divided parallel with the corneal margin at about three or four millimetres from it; beneath the flap thus formed the knife is passed through the sclerocornea into the anterior chamber.[2] Prolapse of the iris into the wound is the complication chiefly to be feared after a sclerotomy: it is avoided by the use of a myotic, careful bandaging, and stillness on the part of the patient. Sclerotomy, like iridectomy, may sometimes be advantageously preceded by scleral puncture.

Under the name of *combined sclerotomy*,[3] De Wecker has lately described a modified operation designed to obtain the advantage of an iridectomy without excision of the iris-segment: a large scleral incision with detachment of the corresponding portion of the iris at its base. After full use of eserine and brief use of cocaine, a scleral incision is made with a broad lance knife; the aqueous is evacuated slowly; the iris is seized with delicate iridectomy forceps close to its periphery and gently dragged towards the pupil. When hemorrhage indicates the detachment of the iris-base the forceps are opened and so withdrawn. The separation of the blades during withdrawal permits escape of blood and prevents extrusion of the detached iris. Eserine is instilled. The operation is said to be easy to perform and unattended by danger.

Other Substitutes for Iridectomy.—In cases of annular posterior synechia with bulging iris it is sometimes impossible to pass the linear knife between iris and cornea. It may then be passed through and behind the iris instead of in front of it, and will, in cutting out, make an *irido-sclerotomy* which will re-establish communication between posterior and anterior chambers

[1] Chirurgie oculaire, Paris, 1870, p. 207.
[2] Trans. Internat. Ophth. Congress, Edinburgh, 1894, p. 12.
[3] Ibid., p. 343.

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and confer the advantage desired. Quite a small aperture in the iris may suffice to permanently banish the glaucoma. The application of this proceeding to primary glaucoma has been suggested,[1] but for most cases is manifestly inadmissible, in view of the close proximity of the lens-margin and the iris-base.

In another form of irido-sclerotomy, or *sclero-iritomy*,[2] the iris-base is divided by cutting from before backward. The knife is introduced as for a sclerotomy; by a quarter turn it is then placed at right angles with the iris,—the edge towards the iris, the back towards the cornea. The blade is rapidly withdrawn in this position. The effect of this is to divide the iris near its base and to give two angular wounds in the sclera well adapted for filtration. Favorable results are reported; but the method involves obvious danger to the lens, suspensory ligament, and ciliary processes.

Incisions and punctures which divide the corneo-scleral junction in a more or less meridional direction, and thus open the aqueous and vitreous chambers simultaneously, were formerly and are still occasionally practised.[3] In acute glaucoma such an incision is capable of giving immediate relief, and is preferable to a simple paracentesis of the cornea, but is not comparable in safety or certainty of result with a well-made iridectomy.

A proceeding has been lately described by which a small, narrow tongue of conjunctiva is pushed through an incision of corresponding size into the anterior chamber. The object is to establish a minute fistulous opening through which the aqueous humor can permanently drain away.[4]

After-Treatment.—The immediate after-treatment differs in no essential respect from that of other major operations on the eye. The dressing must be applied so as to exercise little pressure. All sudden or straining movement must be carefully guarded against. Quietude and sleep must be insured. Closure of the wound, with re-establishment of the anterior chamber, is often present on the first or second day after the operation, but may be delayed a week or longer. So long as the chamber is empty or the wound obviously leaking, the patient should remain in bed.

When the after-treatment is completed, and the refractive condition of the eye has been tested and corrected with a special regard to the astigmatism which results from the operation, the patient should be carefully instructed as to the avoidance of the various conditions and habits of life which, as already described, are causes of glaucoma, and which may lead to its recurrence.

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[1] Knies, Trans. Heidelberg Ophth. Soc., 1893, p. 118; and Ophthal. Review, 1894, p. 24.

[2] Nicati, Revue Générale d'Opht., January, 1894; and Ophthal. Review, 1894, p. 123.

[3] Hancock, Lancet, February, 1860; Solomon, Med. Times and Gazette, 1861–62; Prichard, British Med. Journ., 1871.

[4] Walker, Trans. Internat. Ophth. Cong., Edinburgh, 1894, p. 315.

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WOUNDS AND INJURIES OF THE EYE-BALL AND ITS APPENDAGES.

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INJURIES of the eyeball may be divided into four groups: I., injuries by contusion; II., injuries by penetration; III., injuries by penetration, with retention of foreign body; and IV., injuries by light, heat, and chemical substances.

I. INJURIES BY CONTUSION.

Eyelids.—Ecchymosis of the eyelids is an effusion of blood into the subcutaneous cellular tissue of the lids, with serous infiltration. This condition, known as black eye, is caused by falls or blows, and may also be induced spontaneously by excessive exertion and severe paroxysms of cough.

The thin skin and loose areolar tissue of the lids allow the extravasated blood to spread rapidly before coagulation takes place, thus producing an extensive discolored area. The discoloration of the lids, which is at first dark blue or purple, changes during the progress of absorption to violet, then to yellowish green, remaining visible for some time as a faint coffee-colored or yellow stain. The effusion of blood is eliminated by liquefaction of the fibrin and absorption by the lymphatics, the granules of blood becoming incorporated in the cell-body of leucocytes and carried off by them into the general circulation. The symptoms presented are the swelling and discoloration appearing immediately or soon after the injury. Swelling of the eyelids causes difficulty in opening the eye. There is a sense of stiffness and heat in the eyelids, with marked tenderness at the site of the contusion.

This condition is treated by the application of cold cloths. The swelling generally disappears in a few days, and the discoloration passes away in a couple of weeks. Occasionally it happens that the blood is not absorbed, and an abscess forms in the eyelid. In such cases an abrasion or breach in the skin, allowing pyogenic germs to enter, will be found.

Extravasation of the blood into the lid occasionally takes place after severe injury to a distant region of the head, notably after fracture of the base of the skull. In these cases ecchymosis appears many hours after the

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injury, and there is little or no œdema of the lids, as the ruptured vessels from which the extravasated blood has gravitated are at a distance from the lids.

Emphysema.—Traumatic ecchymosis is sometimes complicated with the entrance of air into the palpebral connective tissue. The ingress of air is made possible by communication with some one of the air-cells which bound the orbit. Emphysema is a sign of fracture of the orbital walls. The fracture causing the emphysema may be situated at some distance from the point of contusion.

In this class of cases we find the lids uniformly swollen and immovable, while the skin becomes tense and shiny. There is little tenderness, and the swelling does not pit on pressure, but resumes its rounded form as soon as the finger is removed. On forced expiration, as in sneezing or blowing the nose, the swelling increases in area. On palpating alternately with the tips of the fingers, crepitation is felt (emphysematous crackling).

The diagnosis is based on these signs, on the history of injury, and on the sudden appearance of the emphysematous swelling or its marked increase after expiration with closed mouth and nose.

The treatment consists in the application of a pressure-bandage, the patient being instructed to abstain from blowing the nose.

Contusion of the region of the supra-orbital margin occasionally produces a clean-cut wound, looking like the result of injury with a cutting instrument. In these cases the traumatic agent is the sharp ridge of bone forming the orbital edge, against which the soft tissues are violently forced and split. Abscess-formation is a frequent complication. At times the skin is intact and the injury periosteal.

The open wound should be carefully cleansed and bandaged. Sutures may be applied later, when we know that the wound is not infected.

Eyeball.—The eye is an elastic capsule. Struck by blunt force, it may be compressed from before backward, shortened in its antero-posterior diameter and lengthened in its vertical one. Every membrane of the eye is thus subjected to great strain in its frontal planes, and may be seriously injured. The traumatisms of the eye due to contusions are prefigured in the anatomy of the organ, and present clinically the ever-recurring pictures of mydriasis, hyphæmia, iridodialysis, dislocation of the lens, and rupture of the globe.

One or several of these conditions may be seen in the injured eye. In my clinic in the New York Eye and Ear Infirmary I recently saw a young girl who, after a blow upon the eye with a fist, exhibited a mydriasis and iridodialysis, a subluxation of the lens, and a rupture of the chorioid.

Contusion of the conjunctiva may be followed by injection and infiltration of this membrane, rupture of the conjunctival vessels, subconjunctival ecchymosis, and laceration of tissue.

Direct contusion of the cornea generally produces an abrasion of the epithelium. The injury is followed by circumcorneal injection, lacryma-

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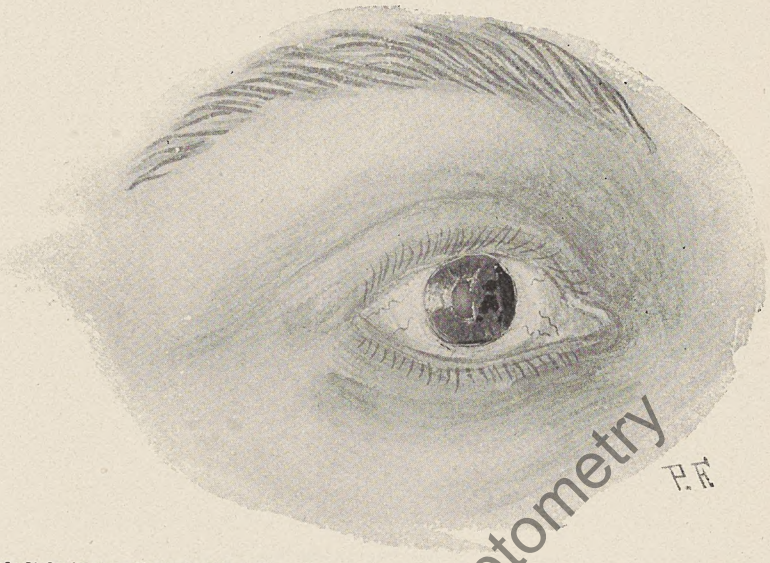
FIG. 1.



Iridodialysis complicated with iritis. From a patient in the New York Eye and Ear Infirmary.
(Drawn by Dr. Percy Fridenberg.)

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tion, photophobia, and pain. The application of a flannel bandage steadies the eye, relieves the pain, and favors epithelial repair.

Contusion of the sclera is a severe traumatism of the eye, and may be complicated with intra-ocular changes.

The sphincter of the iris may be paralyzed by the action of blunt force upon the eye (traumatic mydriasis). The paralysis often affects only some parts of the sphincter muscle, and the resulting dilatation of the pupil is therefore irregular. It is assumed that radiating lacerations in the sphincter are the cause of this iridoplegia. Coincident with this paralysis of the sphincter of the iris we may find a paralysis of accommodation (traumatic cycloplegia). Both conditions are frequently attended with effusion of blood into the anterior chamber. Rest in a recumbent position and cold applications should be recommended in the beginning. Later, when the blood has been absorbed and no iritis has set in, pilocarpine may be instilled. Iridoplegia and cycloplegia from traumatism are generally permanent conditions.

Hyphæmia, an effusion of blood into the anterior chamber, is commonly observed after severe injuries of the iris. It is always present in iridodialysis. At times the blood comes from the ciliary body or the canal of Schlemm. The hemorrhage is usually free, and may fill the anterior chamber. The character of the injury is often concealed by the effusion of blood, and can be known only after its absorption.

Detachment of the iris from the ciliary body (iridodialysis) is of common occurrence after contusion of the globe. The mechanical force flattens the cornea, expands the circumference of the ciliary ring, and subjects the peripheric portion of the iris to great strain. This is still further increased by the backward rush of the aqueous with an impetus derived from the blow. The result is the separation at the point of least resistance at the periphery of the iris. The pupillary portion of the iris is supported by the lens, and does not tend to tear so easily.

The iridodialysis may be small, large, multiple, or complete. When the entire iris is separated from its ciliary attachment, we have a condition named irideremia. The hemorrhage into the anterior chamber is copious at the time of injury, and may recur at later periods in restless patients and at times after the instillation of atropine.

According to Wintersteiner,[1] the profuse hemorrhage into the anterior chamber in cases of iridodialysis is due to rupture of the circulus arteriosus iridis major and of the canal of Schlemm. Ordinary iridectomy is rarely complicated with much hemorrhage, because these blood-channels are not opened in the operation. The defect at the ciliary margin acts optically as an eccentric pupil. It appears as a black crescent or as a segment, and transmits light from and to the interior.

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[1] Beiträge zur pathologischen Anatomie: traumatischen Aniridie und Iridodialyse, Archiv für Ophthalmologie, Bd. ii. S. 1.

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With the ophthalmoscope we see the red reflex of the fundus through the peripheric pupil. The equator of the lens appears as a dark curved line. In case the separation is extensive, the ciliary processes may be seen with oblique illumination.

Iridodialysis is generally a permanent lesion. Occasionally a reattachment of the iris to the ciliary body occurs. Berry [1] mentions such a favorable result from the records of the Dublin Eye Hospital. In the winter of 1895 I had the opportunity of observing an equally fortunate issue in a case of iridodialysis. The patient was admitted to the Mount Sinai Hospital with a small separation at the upper periphery of the iris, complicated with hyphæmia. Under the application of iced compresses, the blood was absorbed within a few days, and the iridodialysis disappeared in the course of two weeks.

In view of the tendency to the recurrence of bleeding, the treatment by rest and iced cloths is applicable to every case of iridodialysis. Occasionally iritis supervenes and requires the use of atropine. The accompanying drawing records such an event. The patient had sustained an injury resulting in a dialysis at the nasal side, and followed by severe inflammation of the iris, with the formation of posterior synechiæ.

A remarkable result of contusion of the eyeball is retroversion of the iris. The condition is either partial or complete. Von Ammon is generally mentioned as having been the first to observe it and demonstrate it anatomically.[2]

Von Ammon, however, yields the priority to J. A. Schmidt.[3]

Inasmuch as laceration of the zonule, partial or complete dislocation of the lens, and detachment of the vitreous are requisite pre-existing conditions for its occurrence, the injury can be produced only by very severe compression of the eyeball. Partial retroversion may be mistaken at the first glance for either an operative or a congenital coloboma; complete retroversion for traumatic aniridia. The differential diagnosis is made by the inspection of the ciliary region. In the various conditions mentioned the ciliary processes are visible by oblique illumination; in retroversion they are covered by the iris, and are therefore invisible.

Traumatic cataract by contusion may present itself as an uncomplicated and comparatively simple condition, or it may be observed in combination with other injuries. The opacity of the lens generally follows the action of the aqueous humor which passes through the ruptured capsule. In the great majority of cases the principal etiological factor is the laceration of the lens-capsule.

The flattening of the cornea at the moment of injury, the coincident in-

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[1] George A. Berry, Diseases of the Eye.

[2] Das Verschwinden der Iris durch Einsenkung, Archiv für Ophthalmologie, Band i. Heft 11, 1854, S. 119.

[3] Ueber eine Art des Unsichtbarwerdens der Iris, Schmidt und Himly, Ophthalmologische Bibliothek, Band cxi., 1804, S. 171.

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crease in the circumference of the ciliary ring, producing sudden and forcible tension of the zonule at its attachment to the lens-capsule, probably constitute the mechanism of rupture. A. Lenz,[1] however, thinks that the momentary compression of the lens in the antero-posterior direction, followed by increase of intra-capsular pressure, causes the rupture of the capsule.

Opacity of the lens caused by simple concussion, without tear of the capsule, is probably less frequent.

In two cases of cataract following contusion, Lawford[2] made an examination of the enucleated eyes and found laceration of the posterior lens-capsule. The posterior capsule, on account of its greater delicacy, probably tears more easily than the anterior. In case the cataract has matured, simple extraction may be performed.

Traumatic Dislocation of the Lens.—After severe contusion of the eye, with or without rupture of the globe, the lens may be forced out of its normal position. The displacement is attended by rupture of the zonule, and may be complicated by other serious injury to the eye. Indirect dislocation after contusion of the globe, without rupture, may be explained by the compression of the eyeball in its antero-posterior diameter with a corresponding increase in the equatorial diameter at the moment of injury. By this change of form the circumference of the ciliary ring and the attachment of the zonule is widened, and a sudden strain, tending to rupture it by stretching, is put on the zonule.

In addition to this factor, we must consider the agency of the contusion, which imparts more or less violent motion to the fluid contents of the globe. The backward rush of the aqueous humor may produce an extensive excursion of the lens, which is heavier than the surrounding media and but loosely suspended. The degree of force necessary to dislocate the lens is variable. The zonule is elastic, and when gradually stretched will allow an axial displacement of the lens of several millimetres, as is seen after the aqueous humor has escaped in the operation of paracentesis.

A sudden sharp blow may cause dislocation. A predisposition to rupture is found when the zonule is very lax or delicate, as in cases of high degree of myopia, fluid vitreous, or staphylomatous eyes. In some of these cases the lens possesses a certain degree of free motility, so that the slightest exciting cause, as an accidental tap on the eye or the commotion produced by sneezing or coughing, is sufficient to induce dislocation.

In some cases of rupture of the globe the lens may be not only dislocated, but forced into the scleral wound or expelled from the eye. If the conjunctiva has not been lacerated at the point of exit of the lens, the latter may be directly beneath it (subconjunctival luxation), presenting itself as a globular mass of pale amber color. Subluxation of the lens is partial dislocation or inclination of the lens-surface without displacement. In

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[1] Hirschberg, Centralblatt für praktische Augenheilkunde, 1897, S. 15.
[2] Ibid., 1887, S. 462.

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such cases the zonule is only partially ruptured. In cases of total dislocation the lens may be forced backward into the vitreous humor, where it settles by gravity, or pushed forward into or through the pupillary space.

Although the lens may be incarcerated in the pupillary area by contraction of the sphincter of the iris, we more frequently find it in the vitreous humor or directly behind the iris or in front of it in the anterior chamber. Here the lens may become adherent to the posterior surface of the cornea. While still mobile, the lens in some cases may be brought from the vitreous into the pupillary space or into the anterior chamber by inclining or nodding the head.

The refractive state of the eye varies according to the position of the lens.

The objective symptoms consist in the absence of the lens from its normal position. They vary somewhat with the degree of dislocation and the state of transparency at the time of injury. In complete dislocation we find the pupil black and lustrous. The contrast, especially in adults, with the other eye may be marked, inasmuch as the faint gray reflex from the surface of the normal lens is missing, as are also the light-reflexes, which are normally obtained from both surfaces. The pupil is usually dilated, at times irregularly. The anterior chamber is deep throughout, or may be shallow at one point from pressure on the iris by the dislocated lens. The iris is no longer supported by the lens, and shows marked tremulousness and quivering visible with movements of the eye or head (iridodonesis).

In case the lens is situated in the vitreous humor, it may be detected by oblique illumination or transmitted light. The periphery of the lens in the vitreous appears dark by transmitted light; in the anterior chamber it assumes a bright golden-yellow tint by direct light. When part of the pupillary space is occupied by the lens the ophthalmoscopic appearance is characteristic. The equator of the lens appears as a dark crescentic line, on one side of which details of the fundus are visible without a convex glass.

In dislocation into the anterior chamber, the lens, being directly illuminated, appears of a yellow-amber color; its periphery looks like a golden-yellow line or thread. When dislocation of a cataractous lens takes place, its discovery, if in the vitreous humor, is easily made. In case its periphery projects into the pupillary space the opaque mass is seen as a crescent, while the rest of the pupil is clear.

In partial dislocation some rays pass through an aphakial pupil, while others are refracted by the peripheric portions of the lens and reach a different portion of the retina, causing monocular diplopia. The dislocated lens tends to assume a globular form, with an increase in its refractive power. Astigmatism may be caused by an inclination of the lens-surfaces. The power of accommodation is, of course, abolished. Dislocation of the cataractous lens is followed by immediate improvement in vision. It is practically a traumatic removal of cataract analogous to the old operation

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of depression. If the capsule is intact, the dislocated lens may remain in the vitreous, there becoming opaque. If the lens remain in the anterior chamber, the cataract develops speedily. The swelling of the lens in the stage of imbibition may block up the iris-angle and produce an acute attack of glaucoma. The dislocated lens may be a constant source of irritation of the iris or the ciliary body, and produce irido-cyclitis or increased tension.

In case all reaction after the injury has subsided, and the presence of the lens in the vitreous does not produce any irritation, it is unnecessary to attempt extraction. A dislocated lens may remain for years in the vitreous without giving rise to any untoward symptoms. This was frequently observed in former years after the operation of depression. Certainly we would hesitate to extract a lens which had been successfully depressed and produced no symptoms. The presence of a dislocated lens in the anterior chamber may be a source of constant irritation and a menace to the integrity of the eye. Removal of the lens is then indicated. The operation is rendered difficult by the tendency of the lens to escape into the vitreous during our manipulations when the patient is recumbent. The operation is simpler in case the lens adheres to the cornea, although some force may be necessary to free it.

The question of extracting a lens dislocated into the vitreous presents itself when glaucomatous tension or reactive inflammation has followed its presence. In the first case, we find that on making our incision at the sclero-corneal margin, the vitreous, which is often fluid, prolapses with some force. If the lens does not present, it is unadvisable to attempt any manipulation for its removal.

Although the immediate results in some of the published cases of extraction are favorable, the termination is generally bad, owing to retinal detachment which so frequently follows excessive loss of vitreous. For the relief of the glaucomatous tension the escape of some vitreous through a small sclero-corneal section is generally sufficient to reduce the tension, and there is little to be gained by attempting to extract an invisible lens. Only in cases of beginning irido-cyclitis it may be absolutely necessary to attempt to extract the dislocated lens which is free in the vitreous. In case of rupture of the globe with subconjunctival luxation the lens may be freed by an incision in the conjunctiva.

So-called serous cysts of the iris, with hemorrhage into the iris-angle, may develop after blunt injury. These cysts generally appear as semi-transparent, grayish-white, globular growths protruding from the periphery of the anterior chamber. They are intimately adherent to the posterior surface of the cornea and to the iris, and it is difficult to remove them *in toto*. According to the results of investigations by Eversbusch,[1] they are in reality sacculations of the ligamentum pectinatum, or inversions of

[1] O. Eversbusch, Beiträge zur Genese der serösen Iriscysten, 1882.

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the iris-angle with hernial protrusion into the anterior chamber. They are more frequently seen after penetrating injuries than after contusion.

Hemorrhage into the vitreous may take place after contusion without rupture of the globe. Retinal hemorrhage or detachment with diminished or absent light-perception, faulty projection, or loss of part of the visual field, may complicate the case and interfere seriously with the recovery of vision. Opacities in the vitreous resulting from hemorrhage may be absorbed by proper treatment, the main features of which consist in rest in the dark, abstraction of blood from the temple, warm applications to the eye, and the internal administration of the iodides or salicylate of sodium.

Rupture of the Chorioid.—Solution of continuity of the chorioid may be found either as a simple condition or as a complication of iridodialysis, dislocation of the lens, and other results of indirect injury. The rupture rarely involves the retina. The sclera is always intact at the point of rupture of the chorioid.

Immediately after the injury, examination is rendered difficult by the irritated condition of the eye, which may cause photophobia and lacrymation. The details of the chorioidal rupture are obscured by hemorrhage and by œdema and cloudiness of the retina. In recent cases chorioidal ruptures appear as yellowish-white streaks, which are usually narrow in proportion to their length. The edges may be yellowish red from effused blood. The rupture may be fissured by smaller secondary rents running at right angles to its course, or it may be forked, branching into a number of secondary divisions at one or at both ends. The retinal vessels may be traced over the ruptured chorioid, except in case the tear has involved the retina also.

The most common form of rupture is that of a narrow band, forming an arc of a circle, which is frequently concentric with the optic papilla. The ruptures may be multiple, appearing in some cases as concentric streaks at increasing distances from the papilla, the smallest streak being the nearest to the disk. At a later stage the yellowish tinge of the rupture changes to white as the remaining shreds of chorioid become atrophic and allow the bluish-white sclera to become uncovered. The edges of the rent may show an accumulation of pigment. Interference with vision varies in accordance with the position of the rupture.

Apart from rupture of the chorioid, other changes may occur in this membrane as a result of contusion. Hemorrhage of the chorioid, followed by the formation of atrophic patches, has been described. Siegrist, who observed areas of partial chorioidal atrophy in the vicinity of the disk in several cases of contusion, ascribes these alterations to rupture of the short ciliary arteries.

Hemorrhage into the chorioid and retina may produce extensive changes in these membranes and impair the sight materially if the macular region is affected. The accompanying sketch was kindly placed at my disposal by Dr. C. A. Oliver, of Philadelphia. The patient, a boy, was struck in

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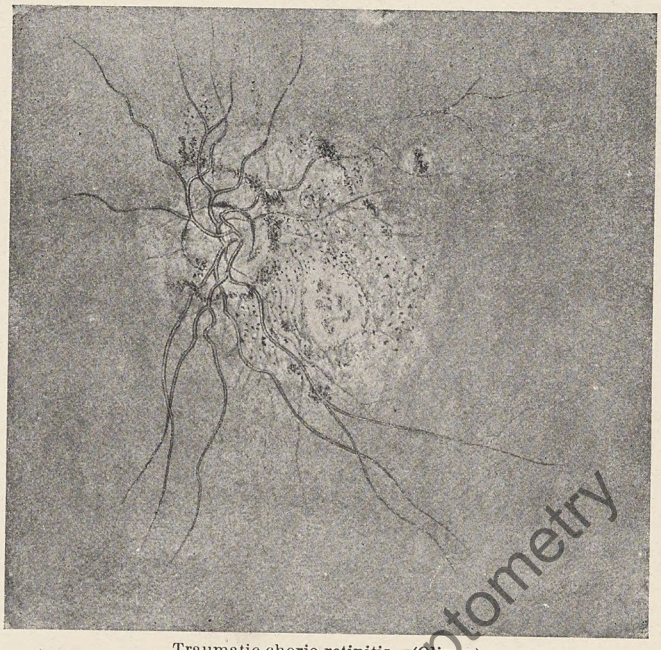
FIG. 4.



Traumatic chorio-retinitis. (Oliver)

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the eye with a stone. Shortly after the injury Dr. Oliver was able to see both chorioidal and retinal hemorrhages. Later the pigmentary and atrophic changes shown in the figure from a sketch by Miss Margaretta Washington developed. Sight was reduced to 2/40 of normal.

Chorioidal injuries are treated on general principles like other severe contusions of the eye. The injured eye is protected from strong light and kept at rest in a moderately darkened room. The use of iced cloths, the application of leeches to the temple, and the instillation of atropine may be indicated to allay pain and check inflammation.

Detachment of the chorioid, induced by hemorrhage into the perichorioidal space, occurs at times as a result of injury by blunt force. The detachment is generally associated with other grave lesions of the eye and with opacities of the refractive media. This is the reason why but little mention is made of the ophthalmoscopic appearance of traumatic detachment of the chorioid. Anatomically it has been demonstrated. At the April meeting (1897) of the Ophthalmic Section of the New York Academy of Medicine, Dr. Arnold H. Knapp exhibited an eyeball which had been gouged and ruptured in a brawl. The interesting feature of the demonstration was the complete detachment of the chorioid by hemorrhage into the perichorioidal space.

CONTUSION OF THE RETINA (COMMOTIO RETINÆ).

Blows on the eye occasion at times a considerable impairment of vision, and the ophthalmoscope shows an area of retinal cloudiness. The affected region appears pale gray in the beginning, becomes intensely white in about twenty-four hours, and gradually clears up after the third day. Associated with these retinal changes we find an episcleral injection and a contraction of the pupil that is not easily overcome by atropine. The disturbance of vision is transitory. A few days after the disappearance of the retinal cloudiness vision is generally restored to its former acuteness. Berlin, who was the first to observe this condition, ascribes it to œdema of the retina induced by hemorrhage into the chorioid. R. Denig[1] produced experimentally the clinical picture of commotio retinæ in rabbits, and demonstrated with the microscope that in these animals the retinal cloudiness is due to the entrance of the vitreous into the layer of nerve-fibres of the retina. He also found a transudation in the layer of rods and cones, and thinks that this is caused by temporary paralytic dilatation of the chorioidal and retinal vessels.

RUPTURE OF THE GLOBE.

In case the contusion is so violent that the requisite compensating distention in the vertical diameter exceeds the elasticity of the eye, a rupture of the whole thickness of the sclera takes place. Such a rupture occurs principally in the anterior sections of the eye. It has, however, been found more posteriorly also, even in the vicinity of the optic nerve. Generally

[1] Paper read before the Ophthalmic Section of the New York Academy of Medicine, April, 1897.

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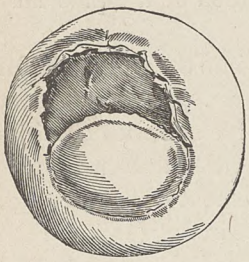
the scleral rupture is situated at a distance of two or three millimetres from the limbus, running parallel to the corneal edge along the canal of Schlemm, the least resistant region of the eyeball. The cornea is partially encircled by the rent, and owing to its greater elasticity escapes injury. The opening in the sclera most frequently lies near the upper and inner circumference of the cornea, and in a region where the iris and the zonula are inserted. The iris and the lens are therefore often forced under the conjunctiva or are thrust entirely out of the eye in case the conjunctiva is also torn (aphakia and aniridia). In spite of the copious hemorrhage which ensues, the eye is not necessarily lost, and even good sight may be regained, for the scleral rupture situated in front of the ora serrata, where the retina is firmly attached, does not always cause detachment of this membrane. In cases with much escape of vitreous such a detachment generally occurs by subretinal hemorrhage more posteriorly in the eye where the rods and cones are but loosely connected with the pigment epithelium. Not only the iris and the lens may prolapse through the rent, but also the ciliary body, the vitreous, and even the retina. Rupture of the globe is a very severe injury, and by far the greater number of eyes thus damaged are lost. The diagnosis of scleral rupture is easily made, even if the conjunctiva remains intact, for in most instances the black color of parts of the uvea or the round yellowish body of the lens can be seen through the conjunctiva. Posterior ruptures may be recognized by the softness of the globe and the presence of corneal wrinkles. In rare cases the scleral rupture is situated in the equatorial region of the globe and the lens is dislocated into Tenon's capsule. Wadsworth[1] reported one case in 1885, and Schlodtmann[2] another in 1897.

FIG. 5.



Rupture of the globe. (Specimen in the author's collection.)

If the conjunctiva is torn and the iris or the ciliary body, or both these structures, and in addition the vitreous, protrude through the wound, it is advisable to snip off all the prolapsing tissues and approximate the lips of the scleral wound by sutures passed through the conjunctiva. In case the conjunctiva is intact, the injury recent, and the iris and the lens have partially or totally escaped through the rent and are held under the conjunctiva, the eye is placed under better conditions for recovery if the conjunctiva is incised and the prolapsed structures are removed. If we keep the patient in a recumbent position, use iced cloths until reaction has subsided, and later apply a compressive bandage, the copious hemorrhage into the vitreous may be absorbed in the course of time. In case the loss

[1] Wadsworth, Dislocation of the Lens under Tenon's Capsule, American Journal of Ophthalmology, 1885, vol. ii. p. 144.

[2] Walter Schlodtmann, Ueber einen Fall von Luxation der Linse in dem Tenon'schen Raum bei äquatorial gelegenem Scleralriss, Archiv für Ophthalmologie, Bd. xliv. Abth. i.

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of vitreous has been considerable and the destruction of the delicate inner tissues of the eye is extensive, it is preferable to remove the eyeball without much delay. Even after the scleral wound has healed we may be obliged to enucleate the eye if it remains irritable and becomes a source of danger to the other eye.

Panophthalmitis is a frequent complication of scleral rupture.

Fissures and fractures of the orbital walls may occur as a result of contusion. The injury is generally accompanied by hemorrhage under the periosteum or into the orbital cellular tissue. Exophthalmos and ecchymosis of the conjunctiva and of the lids frequently follow this injury. If the inner wall of the orbit is fractured, a communication with one or several of the adjacent pneumatic spaces may take place and produce emphysema of the lids and of the subconjunctival and orbital tissues.

There is a possibility of traumatic emphysema whenever, by the action of mechanical force, the continuity of the tissues separating the orbit from the neighboring cavities is destroyed. The cavities and passages to be considered are the frontal sinuses, the ethmoidal cells, the nasal passages, and the antrum.

Fracture of the bony walls of these spaces may establish a communication with the orbit. The result is that, during respiration, the air has free entrance to the latter cavity and its neighboring parts. The normal expiratory pressure is of itself insufficient to force air into the narrow interstitial spaces of the orbital tissues, but its pressure is at times enormously increased during the act of blowing the nose and in sneezing. At such times the air is forced with some violence into the orbital tissues, inflating them and causing protrusion of the eyeball. From the orbit the air may pass into the subcutaneous cellular tissue of the lids and brow, producing immobility and swelling of these parts.

Fracture of the anterior wall of the frontal sinus gives rise to emphysema of the lids when the mucous membrane is perforated, allowing the air to escape through the wound in the act of blowing the nose. As the necessity for this procedure arises only from time to time, emphysema is, as a rule, not extensive, although exceptional cases of general cutaneous emphysema have been described.

Fracture of the lamina papyracea of the ethmoid, or fissure of its wall by extension of the fracture of the orbit or of the basis cranii, is the most frequent cause of orbital emphysema. Numerous cases have been reported in which a blow on the nose or face has produced fracture with penetration of the mucous membrane and consecutive orbital emphysema.

Fracture of the bony wall of the nose, especially of the lacrymal bone and the nasal processes of the superior maxillary, produces an emphysema which affects the lids principally and primarily. The abnormal communication is generally caused by coincident rupture of some portion of the lacrymal sac or duct, so that air is forced from the nose directly into the perisaccular palpebral tissues when the expiratory pressure is raised.

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The principal signs of traumatic emphysema are the protrusion of the globe and an elastic, painless, non-inflammatory swelling of the lids. On pressure, crackling is generally elicited. If the patient is made to blow with closed mouth and nose, the emphysema increases. Differential diagnosis from inflammatory swelling should cause no difficulty. The history points to a sudden development of the swelling soon after the injury, generally on blowing the nose. There is no pain, the only discomfort being caused by interference with free movements of the lids and, occasionally, limitation of the motility of the eye.

The treatment consists in the application of a pressure-bandage and the removal of the cause of a fresh entrance of air by instructing the patient to avoid blowing the nose. Puncture of the skin is never necessary, as the tension of the contained air does not reach a degree that is high enough to cause any untoward symptoms.

Fracture of the roof of the orbit, as a result of a fall or a blow upon the head, generally involves the walls of the optic canal and produces severe injury of the optic nerve. In the vast majority of cases the lesion is confined to one side, and its special characteristic is the sudden, complete, and permanent loss of sight of the affected eye. Exceptionally both optic canals may be fractured. The anatomical explanation of the blindness is the compression, laceration, or crushing of the optic nerve by the fractured walls of the optic canal. A few instances are recorded in which sight returned either partially or wholly. It is assumed that in these cases the loss of function was due to compression by blood effused into the sheath of the optic nerve, and that the absorption of this blood was followed by improvement in sight. Hemorrhage into the sheath is invariably a sign of fracture. In fifty-four cases of fracture of the optic canal von Hölder found hemorrhage into the sheath forty-two times.

Fracture of the roof of the orbit by contusion must be considered as an extension of a fracture of the base of the skull. The traumatism is generally followed by symptoms of basal fracture,—viz., loss of consciousness, severe headache, bleeding from nose or ear, and occasionally paralysis of some of the cranial nerves. The most striking symptom is the sudden loss of sight. The pupil is dilated, occasionally *ad maximum*, and the iris does not react upon direct light, but contracts consensually. Immediately after the injury the ophthalmoscopic examination is, as a rule, negative. Two or three weeks later the optic nerve shows the characteristic signs of atrophic degeneration.

R. Berlin[1] made public von Hölder's autopsies, and was the first to recognize the pathological condition causing the sudden loss of sight.

Enophthalmos, a condition in which the eye sinks backward into the orbit, occurs at times after severe traumatism. The change in the position

[1] R. Berlin, Ueber Sehstörungen nach Verletzung des Schädels durch Stumpfe-Gewalt: Bericht über die zwölfte Versammlung der Ophthalmologischen Gesellschaft, S. 9, Heidelberg, 1879.

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of the eyeball is easily understood in cases of extensive fracture of the orbital walls. It is then a mechanical displacement of the eyeball. If the floor of the orbit is broken, the eyeball may be dislocated even into the antrum of Highmore. Such an occurrence has been observed by Langenbeck. Schapringer cites the case of Smetius, who detected the missing eyeball in the nasal passages. It is difficult to find an explanation for the cases of traumatic enophthalmos without orbital fracture. Beer[1] attributes the condition to atrophy of the retrobulbar cellular tissue, Gessner[2] thinks that it is due to cicatricial fixation dependant upon retrobulbar changes, Denig[3] believes the cause to be irritation of the trigeminus, while Schapringer[4] says that it is injury of the sympathetic inducing paralysis of Müller's muscle. Schapringer argues that the sinking of the eyeball in fracture of the orbit is really a dislocation, and wishes the term enophthalmos to be reserved for the cases without fracture. The bibliography of traumatic enophthalmos collated by de Schweinitz[5] in 1895 enumerates twenty-seven recorded cases.

Pulsating exophthalmos develops at times after severe contusion of the skull. The pathology of the affection is a basal fissure, with rupture of the internal carotid into the cavernous sinus. The injury is followed by unconsciousness, bleeding from the nose or the ear, headache, and at times paralysis of the abducens or of other cranial nerves. These are typical symptoms of basal fracture. The protrusion of the eye makes its appearance several days or weeks after the traumatism. The lids become much swollen and their veins stand out. The bulbar conjunctiva is generally chemotic. When the hand is placed upon the protruding eye and firm pressure is made, the eye recedes into the orbit, and a pulsation synchronous with that of the radial artery and at times a distinct whir can be felt. On auscultation a loud, continuous blowing sound, with systolic increase, is heard over every point of the head, but it is recognized most distinctly over the affected eye and the corresponding temple. Ophthalmoscopically, we see large, tortuous, pulsating retinal veins, small arteries, and a swollen disk; in short, the picture of choked disk. The pulsation and noise cease when the common carotid artery of the affected side is compressed. Ligature of the artery generally effects a complete cure. Several cases of spontaneous recovery have been reported.

In rare cases both eyeballs protrude: such a case has been described

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[1] On Traumatic Enophthalmos, by Theodore Beer: Archives of Ophthalmology, vol. xxii. p. 98.

[2] Enophthalmos Traumaticus, by Gessner: Knapp's Archives of Ophthalmology, vol. xviii. p. 269.

[3] Enophthalmos Traumaticus, by Denig: Archiv für Augenheilkunde, Bd. xxviii. S. 3.

[4] Beiträge zur Casuistik des Enophthalmos Traumaticus nebst Bemerkungen über die Pathogenese desselben, by Schapringer: Klinische Monatsblätter für Augenheilkunde, 1893, S. 309.

[5] Traumatic Enophthalmos, by G. E. de Schweinitz: Transactions of the American Ophthalmological Society, 1895, p. 386.

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by me.[1] A woman fifty-nine years of age fell down a flight of stairs, striking her head violently. She was picked up in an insensible condition, but recovered consciousness in about two hours. On the fourth day after the accident the patient heard a faint noise in the head, which gradually increased in loudness. At the same time she noticed that her left eye squinted towards the nose (abducens paralysis), followed by a protrusion of the same eye. One day later the right eye began to protrude. On auscultation an intra-cranial *bruit* was heard over every part of the head, but most distinctly over the left temple and corresponding eye. Compression of the left common carotid artery stopped the noise; compression of the right reduced it considerably; while prolonged pressure of the right carotid produced dizziness, faintness, and complete syncope. When the hand was placed over either eyeball, a marked thrill was felt. The left eyeball showed rhythmical pulsations at times; the right eye did not pulsate. Finally the patient became blind. An interesting feature in connection with this case is that by ligation of the left common carotid the totally abolished sight was immediately and permanently restored.

The basal lesion causing the arterio-venous aneurism does not seem to extend far beyond the sphenoid bone and its immediate vicinity, for in the vast majority of cases recovery after ligation of the common carotid takes place, and no impairment of function on the part of the cranial nerves remains.

Paralysis of the ocular muscles appearing after injury of the skull by contusion is not infrequently observed. The most common palsy is that of the abducens. This nerve passes over the apex of the petrous portion of the temporal bone, lies in intimate contact with the bone, and is especially exposed to injury in this region, through which fractures of the base usually extend.

The paralysis of the nerve is either primary or secondary,—that is to say, it occurs either immediately after the injury or some days or weeks later. Primary paralysis is ascribed to direct injury by the fractured bone; secondary paralysis is said to be due to compression of the nerve by hemorrhage, inflammatory exudate, or the formation of callus.

Purtscher[2] presents the subject of traumatic paralysis of the abducens in a very complete manner. He has collected and analyzed forty-six cases from literature. Strikingly large is the number of cases of bilateral abducens paralysis. Both nerves were injured thirteen times. In thirty-seven of the forty-six cases the paralysis was probably the direct consequence of the trauma. In nineteen of these thirty-seven the disturbance persisted, and in nine there was recovery. In eight cases the paralysis was secondary. Purtscher assumes that the seat of the lesion was basal in seventeen cases, nuclear in twelve, basal or nuclear in nine, perhaps fascicular in one,

[1] Archives of Ophthalmology, vol. v., part 1.

[2] O. Purtscher, Traumatic Paralysis of the Abducens Nerve, Archives of Ophthalmology, vol. xxiii., No. 4, p. 301.

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possibly cortical in three, and indefinite in one. The frequency of affection of other cranial nerves in traumatic paralysis of the abducens is shown in Purtscher's table of forty-six cases:

The optic nerve was affected	8 times.
The oculo-motor nerve was affected	3 times.
The trochlear nerve was affected	1 time.
The trigeminal nerve was affected	5 times.
The facial nerve was affected	11 times.
The acoustic nerve was affected	12 times.
The vagus nerve was affected	2 (?)
The hypoglossus nerve was affected	2 (?)

Friedenwald[1] has collected eleven cases of traumatic paralysis, seven of which were unilateral and four bilateral. Adding these to the cases of Purtscher, we have a bibliography of fifty-seven cases.

Panas[2] has demonstrated experimentally on the cadaver that by a lateral pressure of five hundred and twenty kilogrammes it is possible to produce a fracture at the tip of the petrous portion, with fissures running into the tympanic cavity, the body of the sphenoid bone, and the optic canal. This explains why paralysis of the abducens is so frequently associated with paralysis of other cranial nerves, notably with paralysis of the acoustic, of the facial, or of the optic nerve.

Panas draws the following conclusions:

(1) Ocular paralysis as a result of injury of the skull is generally due to fracture of the base.

(2) The absence of a depression in the bones of the roof of the skull does not exclude the existence of a basal fissure.

(3) The nerves which lie in immediate contact with the bones of the skull—*e.g.*, the sixth nerve—are especially exposed to injury.

(4) The pressure which causes the paralysis is due either directly to the fracture or to the effusion of blood or plastic exudation. In the former case the paralysis is primary; in the latter, secondary.

II. INJURIES BY PENETRATION.

Wounds of the lids may be incised, punctured, or lacerated, according to the nature of the traumatic agent. They have the general characteristics of all wounds of the outer envelope, and show the same complications in their original manifestations and in the course of healing. Thus, loss of substance, contusion, sepsis, erysipelas, sloughing, and other untoward processes may affect wounds of the lids, to which a special importance is attached on account of their proximity to so important an organ as the eye, the delicacy of their tissues, and the danger of visible disfigurement by scar.

[1] Harry Friedenwald, Traumatic Paralysis of the Abducens Nerve, Archives of Ophthalmology, vol. xxiii. p. 403.

[2] Panas, Paralysies oculaires motrices par pression latérale du crâne, Transactions of the Eighth International Ophthalmological Congress, Edinburgh, 1894.

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The tissue of the lids is extremely rich in capillaries, its stroma being traversed by numerous bands of unstriped muscular fibre. Hence free hemorrhage and lax gaping are characteristic of wounds of the lids. Exact coaptation of the wound-edges is rarely observed when spontaneous union by natural processes has been awaited. Healing by granulation always leaves a scar, and occasionally produces marked deformity (ectropium, etc.), especially if the cicatrix become adherent to the neighboring periosteum and draw on the lids. *Incised wounds* may involve the integument alone or may extend to the deeper tissues and injure the periosteum and the bony structures.

If the edge of the lid be split the lips of the wound separate in the form of a V, giving rise to a condition of traumatic coloboma of the lid, and in the lower lid allowing the tears to run over the cheek and macerate and excoriate the skin.

The deformity is more marked and perfect coaptation is of still greater importance when the tarsal cartilage has been split. The most favorable prognosis attaches, as regards subsequent disfigurement, to linear incised wounds running parallel to and at some distance from the palpebral fissure. The resulting cicatrix is generally linear, as there is no muscular tension causing the wound to gape, and one of the numerous longitudinal skin-folds usually covers the scar.

The muscular fibres of the levator palpebræ may be severed and allow the upper lid to droop (traumatic ptosis). The cut ends of the muscle should be sought for at once, and, when found, carefully united by sutures, as retraction of the muscle-ends makes it difficult to find them at a later period. In the same way one of the recti muscles may be severed or torn from its insertion.

Punctured wounds are of importance because of the frequency with which they are complicated by penetration of the globe or the orbit and by septic infection. It is of great importance, as indeed in all wounds of the lids, to examine the globe with the utmost care in order to exclude penetration or retention of a foreign body. Punctured wounds are usually inflicted by sharp, slender instruments, and the danger of the point breaking off and remaining in the orbit or in the globe must always be borne in mind. As wounds of this sort are rarely attended by free hemorrhage externally, the danger of septic infection in cases in which the traumatic agent is not clean is very great. This imposes upon us the obligation of considering every punctured wound *a priori* as an infected one, and of taking the necessary steps for thorough cleansing. *Lacerated wounds* of the lids are frequently attended by loss of substance and by displacement of important structures. The tear-ducts are occasionally ruptured or torn off, the inner or outer commissure cut through, or the whole lid may be stripped off.

Such wounds are observed in machinery accidents, or after such complicated injuries as a thrust with a pitchfork or a cow's horn, a fall upon a

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hook, etc. These injuries frequently result in marked disfigurement from irregular cicatricial contraction, and offer a wide field for plastic operations. As in the case of punctured wounds, coincident injury of the globe is not uncommon.

Too much stress cannot be laid on the importance of strict asepsis in treating even the smallest cut or tear of the skin. The vascularity and richness in lymphatics of the lids and orbital tissues predisposed them to septic absorption, and even if the septic process be limited to the wound and its immediate neighborhood, the final healing will be delayed; the perfect coaptation which is possible in simple wounds will never be established after suppuration has ensued. As most wounds are inflicted by unclean instruments, and are frequently subjected to further contamination before the surgeon sees them, our procedures must be so modified as to prevent infection or to check a septic process already begun.

We take it for granted that all wounds are septic that have been inflicted by instruments such as a butcher's knife, a rusty hook or nail, a fork or finger-nail, which we know by experience to be generally contaminated, or that show evidence of infection in the presence of foreign matter (hair, dirt, particles of foreign bodies), or in inflammatory swelling or tenderness. These wounds must be carefully freed of all foreign matter, cleansed by irrigation with antiseptic solutions (bichloride of mercury 1 to 1000, carbolic acid two per cent.), and rubbed dry with pads of sterilized gauze or absorbent cotton. A moist aseptic (boric acid, Thiersch's solution) or antiseptic (carbolic acid one per cent.) dressing should then be applied and covered with a layer of gutta-percha tissue (protective, so called) to prevent evaporation and to maintain mild warmth. After the discharge from the wound has diminished and inflammation has ceased, the defect may be allowed to heal by granulation or can be closed by secondary suturing.

In uncomplicated aseptic wounds of the lids our object is to get perfect coaptation of the wound-edges, so that healing by first intention may result. Wounds running at right angles to the course of the orbicularis muscle tend to gape widely, owing to retraction of the cut fibres. Unless special care be taken, the marginal edges of the lid fail to unite, or heal irregularly by granulation, and become permanently notched in the process of cicatricial contraction. The lips of the wound should be approximated by interrupted sutures of fine but strong silk, threaded on small, slender, and well-curved needles, our object in all wounds of visible parts being to avoid disfigurement from stitch-holes and suture-scars. The line of sutures should begin above and end at the free margin in wounds of the upper lid, so that perfect coaptation may result. In wounds of the lower lid the order of suturing must be reversed. It is advisable to have all the sutures in place before knotting them, as imperfect coaptation may then be regulated by varying the tension on the sutures.

The sutures may include the whole thickness of the lid, approximating the cut ends of the tarsal cartilages if necessary. To guard against a

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notching of the lid, an intermarginal suture may be used, which accurately coapts the lips of the wound at the free edge of the lid. For the sake of greater security, this suture may remain *in situ* a day or two after the others have been removed.

In case the lid-wound is complicated by perforation of the tarso-orbital fascia, we may find the orbital fat prolapsing in the wound. This should be snipped off before suturing the lid-wound. After closing the wound the line of sutures should be covered by an aseptic dressing and bandaged. After removal of the sutures the dressing should remain in place a day or two, to guard against possible injury to the young scar in such manipulations as rubbing or washing the eye.

Wounds of the cornea may vary in extent from a superficial defect of epithelium to a perforating cut which traverses the entire thickness of the membrane. Septic infection in the first case and prolapse of iris or vitreous into the wound are not infrequent complications.

Even slight injuries inflicted with unclean instruments—such as a finger-nail, a hair-pin, or a rusty hook—may be followed by a corneal ulcer or by abscess, with danger of sloughing and of panophthalmitis. The cases in which the traumatic agent penetrates into the vitreous, carrying septic matter with it, are especially unfavorable, and generally end in purulent intra-ocular processes. Perforating wounds of the cornea and sclera may be complicated by prolapse of iris or vitreous and by dislocation of the lens or traumatic cataract. Wounds of the sclero-corneal margin are especially dangerous on account of injury to the ciliary body and consequent sympathetic processes, and because of the frequency of complication with prolapse.

Uncomplicated corneal wounds require no operative interference. The surface of the globe should be irrigated with boric acid, a drop of atropine instilled if there is much irritation or any danger of iritis, and a pressure-bandage applied. Local abstraction of blood from the temple is indicated in case of great pain or hyperæmia of the iris. In perforating scleral wounds, a number of sutures may be passed through the conjunctiva after disinfecting, and a pressure-bandage applied to give the eye complete rest and hasten primary union. By this means the lips of the scleral wound are sufficiently approximated. If the iris has prolapsed, the protruding portion should be snipped off with scissors, and if we have reason to suspect infection, the actual cautery should be applied to the stump. Where infection of a prolapsed or incarcerated iris has already taken place, actual cauterization frequently checks the process.

The course of these cases depends on the number and severity of the complications. Coincident traumatism of chorioid, vitreous, or retina of course increases the danger. Occasionally the injury is so severe that the eye is lost at once, while at times insidious irido-chorioiditis results or sight is lost from detachment of the retina long after the injury. Fissure of the iris or loss of substance may be observed after penetrating wounds, or the

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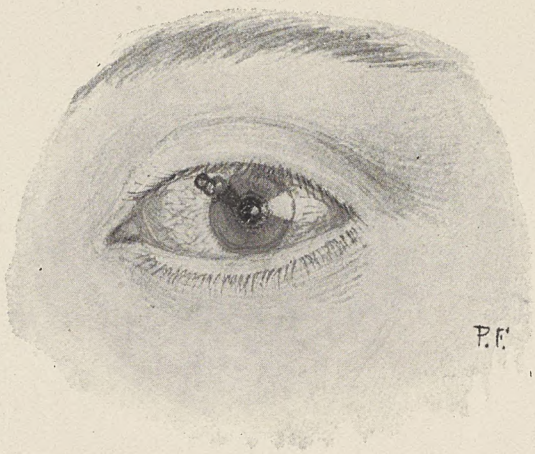
FIG. 6.



Perforating wound of sclero-corneal margin with prolapse of the iris. From a patient in the New York Eye and Ear Infirmary. (Drawn by Dr. Percy Fridenberg.)

FIG. 7.

Dialysis and prolapse of the iris after a penetrating wound of the eye. From a patient in the New York Eye and Ear Infirmary. (Drawn by Dr. Percy Fridenberg.)

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iris may be detached from the ciliary body in part (iridodialysis), or entirely torn out of the eye (aniridia).

The formation of a cyst in the iris-angle is at times observed after penetrating wounds of the eye. The pathology of the cyst is, according to Eversbusch, the inversion of the ligamentum pectinatum and of the endothelial lining of the iris-angle and the accumulation of aqueous humor in the pouch thus formed. The cyst attains a large size, is at times lobulated, and may produce cyclitic irritation and glaucomatous tension. It is a difficult task to remove such a cyst *in toto*. Generally it is excised in part only, and recurs within a short time.

Another form of serous cyst originating at times, after a perforating wound of the eye, is found in the parenchyma of the iris. The membrane is thereby split into two layers, an anterior muscular and a posterior uveal layer. The microscopic examination shows that the cells lining the inner cyst-wall have an endothelial character and can be derived neither from the epithelium of the cornea nor from that of the hair-follicles. The parenchymatous cyst of the iris is globular in shape, has its outline well circumscribed, and can be removed with the part of the iris from which it grows.

The drawing represents a cyst that developed in the parenchyma of the iris after a perforating injury, causing prolapse of iris and cataract. The cyst was wholly removed, and did not return.

The lens-capsule may be pierced or torn by the traumatic agent. The rent may be in the zonule or situated so far peripherally as to be hidden by the iris. After perforation of the zonule near its attachment to the lens-capsule the opacity may show a peculiar star-form situated in the posterior layers of the cortex. In case the perforation is speedily closed, the opacity resulting from imbibition of aqueous may remain circumscribed. Generally the whole lens becomes cataractous, and during the process of imbibition masses of opaque lens-fibres extrude through the rent in the capsule into the anterior chamber. These masses may become entirely absorbed, or their rapid accumulation at the iris-angle may interfere with filtration and give rise to an attack of acute glaucoma. Continued pressure on the iris may be followed by iritis or irido-cyclitis. Such complications indicate the evacuation of the lens-masses, which is best accomplished by linear extraction with iridectomy. It is often impossible to remove all the lens-matter. What remains is generally absorbed spontaneously, although a secondary operation may be necessary.

Dislocation of the lens after penetrating wounds is a severe injury. The *lens* is directly forced out of its position, and may be extruded from the eye, together with iris and vitreous. In such cases the eye is often lost.

The *chorioid* and the *retina* may be directly injured in case of penetrating wound. These injuries are generally severe, and may be complicated by infection.

The *orbit* may be penetrated by the traumatic agent. The result may

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be free hemorrhage behind the globe, infection of the orbital tissues, purulent inflammation, and abscess-formation.

The *periosteum* may be injured or the bony wall of the orbit perforated or fractured, and emphysema of orbit and lid produced. The *optic nerve* may be cut or severed by the traumatic agent, and other nerves and muscles of the eye may be injured.

In case of such complications there is limitation of motility corresponding to paralysis of one or more muscles. As atrophy becomes complete even in partial division of the nerve, sight is often permanently lost.

III. INJURIES BY PENETRATION, WITH RETENTION OF THE FOREIGN BODY.

Injuries to the eye with retention of the traumatic agent are of great importance. The frequency of their occurrence and the severity of the processes to which they may give rise make it imperative for us to be conversant with the clinical aspect of these cases and the procedures for their treatment.

The diagnosis is based principally on the objective manifestations, while the history of the injury and the subjective symptoms are of great assistance.

The history is that of injury by a comparatively small body, usually moving at a high rate of speed. Large bodies lacerate the globe more extensively, inflicting a large wound, through which they are withdrawn or escape spontaneously. Slowly moving bodies do not tend to penetrate the globe, and generally produce injuries by contusion or concussion only. The most common foreign bodies are small chips of metal (steel and iron, less often lead and copper), fragments of stone or glass, and splinters of wood. The industrial population (blacksmiths, engineers, metal-workers, machinists, factory-hands) furnishes a large contingent of these cases. The subjective symptoms are frequently quite out of proportion to the gravity of the injury. The foreign body may cause a sense of irritation ranging from slight itching to severe ciliary pain. The pain may be entirely wanting, and slight photophobia be the only symptom. The vision is usually impaired, especially in recent cases. This may be due to the general irritative state of the eye, to lacrymation and photophobia, or to cloudiness of the media, or it may assume the form of contraction of the visual field or of scotoma. The scotoma may be due directly to the presence of the foreign body, or to pathological changes in chorioid and retina (such as hemorrhage or detachment), or to the formation of dense opacities in the vitreous.

The point of entrance of the foreign body is generally situated in the cornea; less frequently it is in the sclera. The small wound or cicatrix can usually be discovered by oblique illumination. Where the foreign body itself cannot be detected, its course may be traced by the evidence of injury produced in its path.

The iris may show a small perforation or a synechia following localized inflammation. In case the foreign body has penetrated the lens we find an

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opacity which in the beginning is circumscribed, and may remain so, as in the case of small powder-grains or steel chips. Generally, however, the opacity involves the whole lens and increases the difficulty of diagnosis. In a doubtful case the relations of the iris may be of service as presumptive evidence. From theoretical considerations it is evident that prolapse of iris can take place only when certain conditions are fulfilled. The perforating wound must be large; it must gape freely, allowing the aqueous humor to rush out and carry the iris with it. These conditions are not present in the majority of cases of retained foreign body.

The high rate of speed of these small bodies causes them to enter the eye without producing a large or gaping wound. The wound is, in fact, usually linear; the lips come into contact immediately after the foreign body has penetrated, and there is no loss of aqueous.

Large bodies travelling at a low rate of speed produce a large wound, through which the fluid contents of the globe escape, carrying the iris and vitreous humor in to the perforation. The complication of prolapse of iris would then be *prima facie* evidence against the presence of a small foreign body in the eye.

Retention of foreign bodies in the tissues of the lids is comparatively rare. After explosions small grains of powder or sand are found embedded in the skin of the lids. Splinters of wood, portions of projectiles (lead pellets, bits of shell), or stings of insects may penetrate to greater depths. The excessive heat evolved at the moment of explosion renders the metallic bodies sterile, so that they may remain embedded in the lids indefinitely without producing inflammatory reaction. Septic or irritating bodies soon give rise to suppuration and the formation of an abscess about the foreign body, which may be eliminated spontaneously. Grains of powder or sand may be picked out with a spud or cataract-needle. More voluminous bodies may be freed by an incision. As a curious and rather rare form of injury the incarceration of a hair in the tear-point may be mentioned. During the cutting of the hair a small hair may enter the conjunctival sac and, being carried along to the inner canthus, become engaged in the tear-point. It may project sufficiently to irritate the globe with every motion of the lids, much in the same way as a falsely directed eyelash. In this position a small hair escapes all but the most careful examination.

The cornea may be mechanically irritated by objects varying greatly in size and texture. Small particles of dust, sand, or stone, bits of cinder, or ashes, are blown about by the wind in city streets and are frequently carried into the eye. Wings or antennæ of insects, blades of grass or straw, or the hulls of grain are more frequently found in rural practice, while the arts and manufactures supply special foreign bodies, such as bits of glass and metal, chips or sawdust of wood, horn, bone, or stone, tobacco-dust, oyster-shell, etc. Small bodies which do not enter the eye with much force may be carried along by the fluid in the conjunctival sac and finally washed out of the eye. Sharp splinters of glass or metal may easily penetrate and

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remain fixed in the coats, especially when they strike the eye with force, as in explosion of soda-water bottles or of the bulbs used for the incandescent electric light.

The examination of the cornea requires special thoroughness. Owing to the variegated background presented by the iris, the color of the foreign body offers no contrast, while the bright reflex from the curved surface of the cornea increases the difficulty of detection.

By oblique illumination we may bring out the details of the background, while the surface of the cornea casts its reflex to the side from which the light is thrown, and not towards the eye of the surgeon. The reflection of the window or of some large white object on the cornea may be used to detect abrasions or epithelial defects by their causing a break in the mirrored image. This may also be accomplished by allowing a few drops of a one per cent. solution of fluorescin to flow over the surface of the cornea. The solution affects only a roughened, denuded spot, staining it a light green, and affording a marked contrast to the foreign body.

Treatment consists in the removal of the foreign body. Bits of dust or cinder on the palpebral conjunctiva may be wiped away with a bit of cotton on a probe. Foreign bodies on the cornea may be lifted off, or, if necessary, dug out with a spud. The surface of the eye should then be irrigated with a solution of boric acid, or with some antiseptic if we have reason to suspect infection. If there is hyperæmia of the iris or pain, atropine may be instilled. A bandage should then be applied. Occasionally foreign bodies become buried so deeply in the cornea that an unskilful attempt at removal may force them into the anterior chamber. In this class of cases pressure from behind should be continued during the process of extraction. For this purpose a broad needle is thrust into the anterior chamber from the margin of the cornea, and the point applied to its inner surface directly behind the foreign body.

Chips of iron frequently reach the eye in a glowing state, and their mechanical action is complicated by a burn. The reaction in these cases is marked, and a small portion of the cornea immediately surrounding the foreign body becomes necrotic and sloughs. The iron enters into chemical combination with the albuminoid substance of the cornea and produces a grayish-brown discoloration which closely resembles a foreign body.

The penetration of the cornea and the conjunctiva by the hairs of certain caterpillars furnishes a clinical picture which differs from that caused ordinarily by foreign bodies. The initial symptoms are those observed in other cases of conjunctival and corneal lesions; but the later and characteristic features appear in the third or fourth week, and consist in the formation of small nodules in the bulbar and palpebral conjunctiva. The nodules develop around the hairs of the caterpillar, have the size and color of a millet-seed, are movable over the sclera, and are composed of granulation-tissue with many giant cells. The disease, which has been named ophthalmia nodosa or pseudo-tuberculosa, is protracted even in its mildest

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forms, and may in its graver manifestations lead to the destruction of the eyeball. The hairs may pass through the cornea, reach the iris, and here give rise to the development of nodules with severe inflammatory complications on the part of the uveal tract.

Foreign bodies in the sclera are rare, as they easily rebound from the conjunctival covering, or, on the other hand, have sufficient force to penetrate the globe. Particles of glass entering the eye produce but little reaction in the beginning. In the course of time, however, inflammatory symptoms appear and increase slowly and steadily. Leber thinks that glass is chemically not indifferent. In a case reported by Wagenmann[1] a splinter of glass had passed into the eye and had remained in the anterior chamber six months without producing any signs of irritation. After that time mild inflammatory signs appeared and the cornea grew hazy. This irritation lasted seven months, but improved speedily after the removal of the glass sliver. Wagenmann contends: (1) that the reaction was not mechanical, because the foreign body was fixed in the tissues; (2) that the reaction was chemical, because it appeared so long after the traumatism.

At times an eyelash is carried into the eye in perforating traumatisms and in operations. It causes hardly any reaction, and may be considered as an indifferent foreign body giving rise neither to mechanical nor to chemical irritation.

An epidermoidal cyst may grow in the iris as the result of traumatic transplantation either of the epidermis of the lids or of the epithelium of the cornea or the conjunctiva. It is probable that in these cases glandular tissue is carried into the iris. The epidermoidal cyst appears as a small, white, globular mass that is semi-solid in consistency and contains flat cells both with and without nuclei.

Treatment consists in the excision of the cyst with the iris-tissue from which it grows.

Foreign bodies in the anterior chamber sink to the bottom, where they are detected with difficulty. Localized signs of irritation, as persistent injection at one point of the sclero-corneal margin or a drawing out of the pupillary margin towards the periphery of the anterior chamber, are aids in discovering the foreign body. Occasionally the latter may be covered by an exudate of inflammatory products and appear as a small node of pus or bloody lymph.

Foreign bodies in the iris are comparatively rare. A body striking the eye with such force as to penetrate the cornea or sclera usually retains enough impetus to pierce the delicate tissues of the iris. Chips of metal or of stone are the bodies that are most frequently found. Small bits of stone or steel may remain in the iris for many years without producing inflammatory reaction. Copper is much more dangerous, even when bacteriologically clean, as it gives rise to severe irritation by its chemical reaction. The

[1] Archiv für Ophthalmologie, xl. 5.

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detection of a foreign body in the iris is rarely difficult, although in a fresh case the presence of blood in the anterior chamber and haziness of the cornea from coincident traumatic keratitis may obscure the field. In case the foreign body has carried germs of septic infection with it into the eye, severe inflammation almost invariably results, as the vascular iris offers a particularly good soil for the propagation and transportation of infectious material. Foreign bodies in the iris may be removed by forceps or by the magnet (in case of steel or iron chips) through a corneal section. Excision of the fold of iris enclosing the foreign body is frequently necessary.

Grains of powder or sand blown into the iris by explosions produce marked inflammatory reaction. Extraction of these numerous and minute bodies, which are deeply buried in the tissue of the iris, is impossible, and our treatment must be limited to the iritis and its complications.

The presence of a foreign body in the lens is followed by a localized or general opacity. Operative interference in the beginning is not advisable unless there is some urgent indication for the removal of the lens. The danger of acute glaucoma during the stage of imbibition or of irritative irido-cyclitis should, of course, determine prompt extraction with iridectomy. Otherwise we may wait until cataract is fully formed before attempting extraction.

The presence of a foreign body in the vitreous is always a menace to the integrity of the eye. The prognosis is grave, although exceptionally small aseptic metallic fragments have been observed to remain in the globe for years without giving rise to destructive processes. Although there may be some doubt as to the advisability of attempting removal of the foreign body in every case, the patient should be kept under observation, as reactive inflammation may break out after a long period of quiescence and require prompt action. A foreign body in the vitreous may be hidden by opacities of the media, by hemorrhage into the vitreous, or by an exudate. It may penetrate the retina or may lie on the posterior surface of the iris or on the ciliary body, where it is inaccessible to ophthalmoscopic examination.

Because of the tendency to sink and come into contact with the uvea, early extraction of freely movable bodies is advisable. In the case of iron or steel bodies the use of the magnet is indicated. Operative interference should be deferred only when the site of the foreign body cannot be determined and the eye remains quiescent, or when the foreign body is fixed in the coats of the eye and encapsulation may be expected; but in such cases the patient should be kept under observation.

If the presence of a foreign body is suspected in the interior of the eye, but cannot be demonstrated by the ordinary methods of inspection, other diagnostic measures may be applied, viz.: Asmus's sideroscope; Haab's magnet; Roentgen's rays.

The sideroscope[1] is an instrument for the demonstration of the presence

[1] Das Sideroskop, Edward Asmus, Archiv für Ophthalmologie, Band xl., 1894, S. 280.

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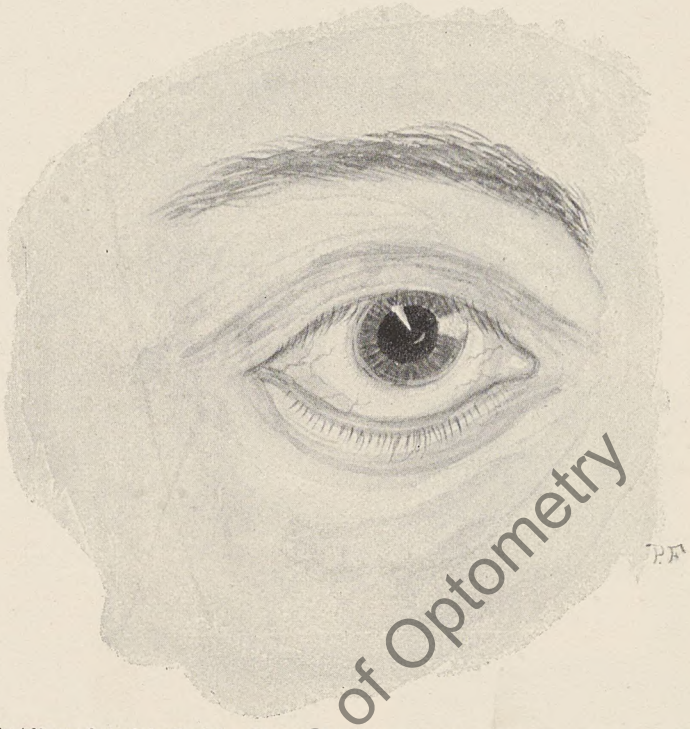
FIG. 10.



Foreign body (sliver of steel) in the iris. Removed by magnet without iridectomy. From a patient in the Mount Sinai Hospital. (Drawn by Dr. Percy Fridenberg.)

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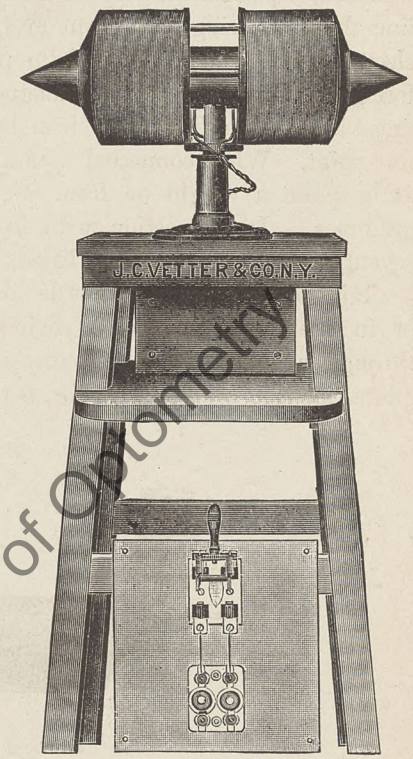
of particles of iron or steel in the interior of the eye. The apparatus consists of a magnetic needle, the deflections of which are shown on a scale and are read by means of an astronomical telescope. In twenty-five successive cases observed at the University Eye Clinic in Breslau in 1893 and 1894, fragments of iron or steel were detected by this instrument. Dr. Thomas R. Pooley,[1] of New York, was the first to employ a magnetic needle for the purpose of determining the presence of steel in the eye. Asmus made Pooley's magnetic needle more available for practical application by the addition of a scale and a telescope.

Haab has constructed a large electro-magnet for the removal of pieces of steel from the interior of the eye. The body of the magnet is a cylinder of soft iron measuring ten centimetres in thickness and sixty centimetres in length. The cylinder is provided at each end with a detachable conical point, and wound with a coil of copper wire weighing fifty-seven kilogrammes. The apparatus is supported on a solid framework of wood, measures one hundred and thirty-five centimetres in height, weighs one hundred and thirty-eight kilogrammes, and can be worked by connection with a dynamo or with the street current. It is a useful instrument both for the diagnosis of the presence of particles of iron and for their removal from the interior of the eye. In injuries with retention of fragments of iron, the approximation of the eye to the instrument causes in many cases, especially when the substance is not too firmly embedded in the fundus, a sensation of acute pain, and reveals the presence and often the exact location of the foreign body. With the aid of Haab's magnet it is, furthermore, possible to draw particles of iron from the vitreous forward into the anterior chamber, and at times through the original or enlarged wound out of the eye. Thus the meridional incision through the sclera and the introduction of the magnet into the vitreous can be avoided.[2]

FIG. 11.



Haab's magnet.

[1] Thomas R. Pooley, On the Detection of the Presence and Location of Steel and Iron Foreign Bodies in the Eye by the Indication of the Magnetic Needle, Archives of Ophthalmology, vol. ix., 1880, S. 219.

[2] O. Haab, Ein neuer Electromagnet zur Entfernung von Eisensplittern aus dem Auge, Beiträge zur Augenheilkunde, Heft xiii. S. 68, 1894.

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The application of the X-ray has been shown to be useful in locating foreign bodies in the interior of the eye. Charles W. Williams, of Boston, reports a case in which by the aid of a skiagraph he recognized the position of a piece of copper in the vitreous. C. F. Clark, H. F. Hansell, G. Oram Ring, G. E. de Schweinitz, and others have in the same manner found fragments of steel in various parts of the eye, and Percy Fridenberg has detected in the orbit the presence of small shot which had passed through the eye. Hirschberg[1] doubts the utility of skiagraphs as guides in the extraction of copper fragments from the interior of the eye. He says that the first condition of success in these operations is the exact diagnosis of the location of the foreign substance. The skiagraph avails nothing; the fragment itself must be seen, or at least the path that leads to it must be in a straight direction.

The magnet was first introduced into ophthalmic practice by McKeown, who used a permanent magnet eight inches long, one inch broad, and one line thick, tapering to a point at both ends. Hirschberg constructed an electro-magnet and formulated the principles for its use in the extraction of foreign bodies. Hirschberg's magnet consists of a closely wrapped coil of fine wire surrounding a soft iron bar, one end of which is drawn out to a fine point. When connected with a zinc-carbon element, this magnet raises at its point a weight of from one hundred to one hundred and twenty grammes. In connection with five cells as much as five hundred and seventy-five grammes can be lifted.

Hirschberg's magnet may be introduced through the original wound, or, in case this is impervious or inconveniently situated, it may be inserted through a section at the sclero-corneal margin, or through a meridional scleral incision near the equator, between two of the recti muscles.

Fig. 12.

Hirschberg's electro-magnet.

A permanent magnet constructed on the principle of the magnetic magazine has been described by me in the *New York Medical Record*, May, 1880. The apparatus consists of a number of steel rods fitted into iron caps, one of which is provided with a conical point of malleable iron. By

[1] Ueber Entfernung von Kupfer-Splittern aus dem Augengrunde, Berliner klinische Wochenschrift, 1897, No. 15.

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contact with a dynamo the apparatus is made magnetic, and remains charged for about one year. The instrument is always ready, and has proved quite useful in the extraction of visible and movable iron particles from the interior of the eye.

FIG. 13.

Gruening's permanent magnet.

The prognosis in cases of retention of iron or steel fragments has become much more favorable since the introduction of the magnet into ophthalmic practice, the great advantage of the procedure lying in the fact that it enables us to extract not only visible iron particles, but also those whose exact position in the interior is a matter of conjecture. The cases in which the foreign body is of non-magnetizable metal are much less frequent than formerly, when injuries from fragments of copper or brass percussion-caps were common. The use of these caps still survives in cheap fire-arms, toy pistols, and detonating railroad signals, so that injuries of this kind occasionally occur.

The cases in which extraction with the electro-magnet has not been followed by recovery are those in which infection had taken place at the time of injury and progressed steadily, or in which the mechanical injury produced by the foreign body or by the introduction of the magnet has been followed by reactive inflammation and destructive purulent processes terminating in panophthalmitis, or by the formation of connective tissue with detachment of the retina or traction on the ciliary body.

The question of further operative interference arises when an attempt at extraction has failed, or when such an attempt is contra-indicated. Failure to extract with the magnet may be due to the fact that the foreign body is fixed in the wall of the globe or is encapsulated in dense membrane, in which case an attempt at extraction with ribbed forceps may be made.

Fragments of copper produce purulent inflammation by chemical action, and should, therefore, be removed speedily. This can be done with ease in case the foreign body is situated in the anterior chamber, on the iris, or in the lens. When the copper fragment has passed into the vitreous humor extraction is more difficult, but should be attempted even if purulent infiltration has set in. Leber[1] has shown that the pus may be the product of chemical action alone, and that, in the absence of other complications, all inflammatory symptoms may disappear after the removal of the copper from the vitreous. Not only is the form of the eye almost invariably preserved, but also some useful sight can be retained in a considerable number

[1] Leber, On Perforating Injuries of the Eye by Morsels of Copper, and on their Treatment, Transactions of the Eighth International Congress, Edinburgh, 1894.

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of cases, and all this, according to Leber, without the risk of sympathetic ophthalmia.

In all cases in which the foreign body is still in the globe our treatment should be guided by the cardinal principle of preserving as long as possible, first, the function of the eye; second, its form; and of deferring enucleation until we are obliged to remove the globe on account of progressive inflammation or danger of sympathetic ophthalmia.

In case the foreign body is encapsulated or fixed in the coats of the eye, its presence may cause absolutely no reaction.

The presence of particles of iron in the interior of the eye affects certain groups of cells, as the epithelium of the ciliary processes, the pars ciliaris retinæ, the retina, and the epithelium of the lens-capsule. Von Hippel[1] names this condition xenogenous siderosis, distinguishing it from hæmatogenous siderosis. The circle of brown spots under the lens-capsule in cases of retention of a fragment of iron in the eye is clinically well known.

According to von Hippel,[2] the lodgement of even an aseptic fragment of iron in the retina may at times lead to degenerative changes that are due to chemical action. He publishes the history and gives an anatomical description of a case in which the entire retina had been transformed into pigmented connective tissue.

That such degenerative changes do not invariably occur is amply proved by clinical observations. The cases in which the foreign body is encapsulated run a more favorable course. I have seen several instances of this.

Fig. 14 shows the ophthalmoscopic picture of a foreign body that was encapsulated in the retina. For many years I observed this case, in which a chip of steel had entered the globe, passing through the lens and producing traumatic cataract. After removal of the cataractous lens the foreign body was seen in the retina, encapsulated in a whitish substance and surrounded by an area in which there were slight chorioidal changes. Until the death of the patient, fifteen years later, this foreign body caused no irritation or interference with vision, while the vitreous remained transparent and free from connective-tissue formation. In this case lenticular opacity prevented an examination of the interior of the eye, and the patient was kept under observation until the cataract had matured. Extraction of the cataract then allowed me to determine the position of the foreign body.

In cases of injury by gunpowder or dynamite explosions, numerous small grains of burnt powder, sand, or infusorial earth, together with steam or gas, are thrown at times with great violence into the eye, peppering the cornea, the iris, and the lens, and sometimes penetrating back to the vitreous. These foreign bodies are so numerous and so small that it would be impossible to remove them were it even advisable to do so. As they are

[1] Siderosis Bulbi and the Relations between Siderosis and Hæmatogenous Pigmentation, E. von Hippel, Archiv für Ophthalmologie, xl. 1.

[2] Ueber Netzhaut-Degeneration durch Eisensplitter, E. von Hippel, Archiv für Ophthalmologie, xlii. 4.

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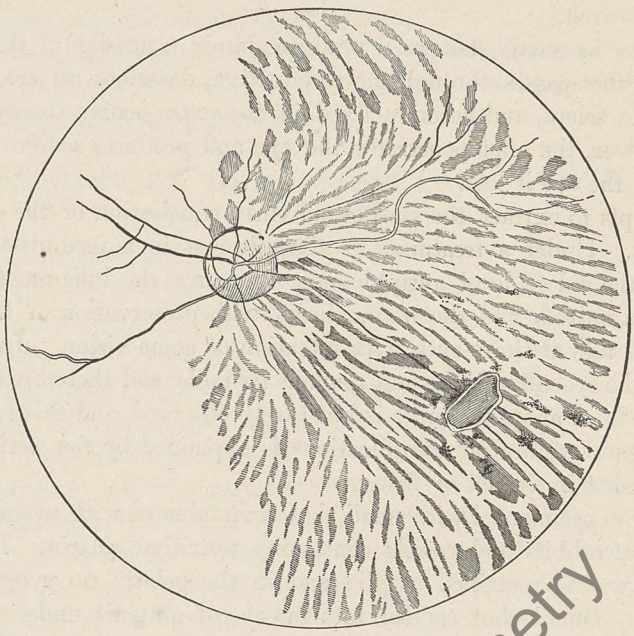
usually aseptic and chemically inert, the reaction produced by them is due to mechanical irritation alone. Thus traumatic cataract is frequently produced, in the course of which glaucomatous symptoms may appear. Our treatment is limited to extraction of the cataractous lens and the relief of the inflammatory processes in iris and cornea.

FIG. 14.



Encapsulated foreign body in the retina. Drawn from a patient in my private practice by Dr. Albert H. Fridenberg.

Foreign Bodies in the Orbit.—A foreign body may lodge in the orbital tissues and give but little evidence of its presence. The traumatic agent, generally a pointed piece of wood, a lead-pencil, or a metallic pen, may break during penetration, and the broken end be retained in the orbital cavity. The external injury is a punctured or small lacerated wound, and gives no clue to the depth and direction of penetration. R. Berlin called attention to the fact that in the moment of penetration the eyeball is apt to rotate in the orbit, so that the wound-canal changes its course. When the eye returns to its original position the wound-canal is occluded by angular deviation. A probe does not enter far through the external wound, and we may be easily misled in our conclusions as to the depth of the penetration. If the eye was open at the moment of injury, the external wound may be situated in the conjunctiva of the globe. At times the foreign body lodges between the globe and the osseous walls and injures no important organ; at times, however, it destroys sight and even life. In its passage it may open the globe, tear the optic nerve, break through the osseous wall, and

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do harm to the brain. It may infect the orbital tissues, and produce suppurative cellulitis, meningitis, and death.

The treatment consists in the extraction of the foreign body through the original wound, if possible; and if not, through either the upper or the lower oculo-palpebral fold. In some instances it may be necessary to resort to osteoplastic resection of the outer orbital wall. Small and aseptic foreign bodies demand an expectant course and do not necessitate an immediate operative search.

Injuries by small shot may produce simple contusion of the eye. At times the shot passes through the conjunctiva, describes an arc of a circle around the sclera, and becomes fixed in the extra-ocular tissues. Generally, however, the shot penetrates the eye and produces severe symptoms. As a rule, the eye is lost.

Attempts to extract the shot or an early enucleation of the eye are not justifiable. Under a treatment of absolute rest in a recumbent position, and with the aid of local antiphlogistic measures, the inflammatory symptoms often subside and the wound heals, with preservation of the form of the eyeball and at times even with retention of some vision. In unfavorable cases the eyeball shrinks and remains irritable, and therefore enucleation is indicated. Panophthalmitis is but rarely observed, and this relative immunity from suppurative complications is explained by the sterilization of the small shot in the act of firing.

Ovio[1] experimented with small shot contaminated with pyogenic germs. He fired at rabbits and vessels containing nutritive gelatin. In the animals the wound remained aseptic, and in the gelatin no pyogenic germs developed. Small shot carried into the eye of animals under aseptic precautions caused no reaction, the eye remaining quiet many months.

Gunshot injuries of the eye are generally associated with severe lesions of the neighboring parts. The brain, the face, or the cranial bones may receive an injury involving the eye. During the Franco-German war (1870–1871) Herman Cohn[2] observed thirty-one cases of gunshot wounds affecting the eye. In two cases the projectile struck the brain, in nine the face, in four the cranial bones, and in sixteen the eye directly.

Cohn enumerates seventy different pathological conditions found in the injured eyes. Among the principal lesions noted are: total destruction of the globe, atrophy of the globe, wounds of the globe with or without retention of foreign substance, iridodialysis, mydriasis, hemorrhage into the vitreous, rupture of the chorioid, detachment of the retina, atrophy of the optic nerve, paresis of the rectus internus muscle, and paresis of the rectus externus muscle. It is easily understood how this variety of lesions is produced. In passing through the osseous walls of the orbit the bullet imparts motion to bone fragments, which, together with the projectile, constitute crushing,

[1] Ovio, Sur la pénétration de grains de plomb dans le bulbe oculaire, Revue générale d'Ophtalmologie, 1895, p. 305; Michel, Jahresbericht der Ophthalmologie, 1895, S. 574.

[2] Die Schussverletzungen des Auges, Herman Cohn.

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lacerating, cutting, and stretching agents. Quite frequently particles of extraneous matter, such as stone, gravel, cloth, parts of eye-glasses, are carried into the eye with the ball, and thus the already grave injury may be rendered more serious by the forcible entrance of foreign bodies. Furthermore, the injury may be complicated with burns of the eye and the surrounding parts. Thus, contusion, penetration with retention of foreign substances, and burns may be associated with gunshot injuries, and the whole gamut of traumatic possibilities may be exhausted.

The treatment of gunshot wounds is conducted on general surgical principles. If the eyeball is destroyed, it is advisable to remove what is left of it, in order to establish a clean wound.

IV. INJURIES BY HEAT, LIGHT, AND CHEMICAL SUBSTANCES.

The destructive action of excessive heat on the delicate tissues of the eye is frequently observed, as the opportunities for accidental injuries of this sort are many in domestic and in industrial life. In the one case we have to deal principally with the injuries caused by hot fluids (boiling water, soup, fat, or lard, etc.) used in the household; in the other with masses which have been subjected to intense heat during the process of manufacture (glowing iron, molten lead, hot pitch and tar). Not a few burns are caused by the glowing ends of cigars and cigarettes, by burning shreds of tobacco blown from pipes in a wind, by the ends of sulphur matches, etc.

Burns of the lids are of importance on account of the frequency of disfigurement produced by cicatricial contraction after extensive destruction of lid-tissue, and of the complication with burns of the surface of the globe. They may be of a high degree, but are usually not extensive, except when in rare cases they form part of a general burn. They are most frequently caused by small incandescent or flaming objects, such as sparks from fireworks, glowing cinders, tips of burning matches, drops of molten metal. The extremely high temperature of these bodies explains the intensity of the burns, as it may allow them to penetrate the entire lid before cooling sufficiently to be arrested in their action. The harmful results of superheated fluids or vapor on the lids is much like that of burns, except that the injury is of a like degree throughout and very extensive. The diffusible nature of the traumatic agent explains this peculiarity, as the fluid flows over the lid or is scattered by splashing. The treatment of burns and scalds of the lid is conducted on the general surgical principles established for such injuries, the details varying with the degree and extent of the burn. Our first step is to prevent infection of the parts and to protect them from the outer air. This is best accomplished by applying a surgical moist dressing, consisting of a loose compress of cheese-cloth or gauze wrung out in a weak antiseptic solution (boric acid three per cent., carbolic acid one per cent.). This is covered with a layer of rubber protective tissue to prevent evaporation and maintain mild warmth. These dressings must be frequently

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changed, and the outer surface of the lids should be irrigated with a solution of boric acid. The advantages of this moist aseptic dressing are numerous. It absorbs the wound secretions perfectly and renders them aseptic, protects the wound from the air and mechanical irritation, prevents drying of the surface by evaporation, increases local temperature, assisting the process of repair and the elimination of necrotic tissue, and has a noticeable anodyne action. In the stage of granulation of burns in which the epidermis has been destroyed, the wet dressing must be slightly modified as the fresh granulations grow through the meshes of the gauze. At each change of dressings these granulations are torn off, producing free hemorrhage and delaying the process of repair. To prevent this, the surface of the granulating burn must be kept from contact with the gauze. This may be accomplished by thickly powdering the wound with bismuth or iodoform, or, better still, by covering it with a layer of rubber tissue. This layer must be specially prepared to prevent retention of wound secretion by cutting it into strips about four times as long as wide. These strips are rendered aseptic, moistened in boric acid or Thiersch's solution, fenestrated with a scissors or a punch, and laid side by side on the wound. The moist gauze may be applied over this layer and removed whenever a change of dressing becomes necessary, without giving rise to pain or disturbing the process of healing. The wound secretions are absorbed through the apertures in the strips, and the burns heal promptly.

Limitation of the inflammatory reaction is of great importance in all injuries of the coverings of the eye, and especially so in burns which are accompanied by much irritation. The condition of cornea and iris should be carefully noted, while that of the inner surface of the lid requires special attention. The symptomatic treatment consists in relief of pain and swelling, which are often severe and may require the administration of anodyne and the application of antiphlogistic remedies. A certain degree of systemic disturbance, with general prostration and, occasionally, febrile reaction, may be observed when the burns are extensive.

The conditions resulting from burns of the lids frequently require operative treatment.

Various degrees of ectropion or of symblepharon may be produced, the former by the adhesion of a cicatrix to underlying bone, from which as a fixed point it draws upon the lids, the latter by a coalescence of the denuded lid with the globe. Symblepharon may be so complete as to preclude any improvement from operation; usually, however, a plastic operation effects a cure of this condition. Transplantation of small islands of epidermis by skin-grafting (Thiersch) onto the granulating surface of a burn frequently hastens the process of healing and diminishes the amount of cicatricial contraction.

Powder burns are complicated by the mechanical action of the powder-grains, which become embedded in the lids, and by the action of the flame in case the explosion has taken place close to the eye, singeing the lashes

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and brow and searing the lids and surface of the globe. The immediate reaction is intense; but, although ulceration or abscess-formation occasionally results, the presence of the powder-grains, as a rule, produces no destruction, and may be tolerated for an indefinite period. Complication of the burn with severe injury by mechanical force is observed when other foreign bodies, such as bits of stone or metal, are violently thrown against the eye by the force of the explosion. These injuries should be treated by careful removal of all foreign bodies under aseptic precautions, cleansing by irrigation of the conjunctival sac, and application of an occlusive bandage. In case of much pain, cold compresses may be applied. If there is a complicating iritis or tenderness in the ciliary region, instillation of atropine, abstraction of blood from the temple, and the use of warm compresses are indicated.

The cornea and sclera are usually affected in all but the most superficial burns of the eye. The injury of the cornea may be very variable in severity. There may be only a localized infiltration of the superficial layers, or the epithelium may be clouded and steamy.

In case of severe burn, the entire thickness of the cornea may be converted into a dense white tissue resembling porcelain.

The apparent absence of inflammation may mislead us in these cases. The cornea is entirely anæsthetic, but there is generally severe intra-ocular pain. The diffuse white opacity is composed of necrosed tissue, which sloughs, allowing perforation of the globe to take place. Even superficial injuries may become serious if infection take place, or the nutrition of the cornea may be greatly disturbed, producing ulceration or abscess of the cornea. The conjunctiva of the globe is generally affected to a marked degree. There is intense injection, and, at times, ecchymosis, while after severe burns the entire conjunctiva may be destroyed and cast off, laying bare the sclera. On these denuded areas granulations arise which may lead to coalescence of the lids with the globe, producing more or less extensive symblepharon.

By the motion of the lids the inflamed and chemotic conjunctiva may be pushed over the cornea and adhere to its surface as a traumatic pterygium.

In case the cornea is not destroyed, a dense opacity (leucoma) generally remains at the site of the burn. The treatment of burns of the surface of the globe consists in disinfection by irrigation with weak antiseptic solution and application of cold compresses. The surface of the globe may be lubricated by dropping a solution of glycerin and rose water into the eye, or a drop of sweet oil to prevent coalescence of the apposing lid-surface.

Prevention of intra-ocular inflammation and extension of the process is of importance. The eye should be irrigated frequently. In case of ciliary pain or hyperæmia of the iris, atropine should be instilled, leeches applied to the temple, and mercury administered, while the patient is kept in bed in a darkened room. During the stage of reaction of deep burns, with

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the formation of sloughs, warm compresses should be applied to assist in the process of casting off the necrotic masses. In a later stage there is usually an irritative condition, with slight conjunctival discharge, for which a 1 to 500 solution of silver nitrate or 1 to 500 zinc sulphate can be used.

Small drops of molten lead are occasionally splashed into the eye, and may produce very little destruction. The rush of tears and the fluid in the conjunctival sac immediately reduce the temperature of the metal, while being themselves partially converted into a thin film of steam which protects the globe from injury. The metal solidifies in cooling, moulding itself to the form of the globe, and may be picked off the eye with little difficulty.

Corrosion of the eyes by concentrated acids or alkalies is more dangerous. The destructive action of strong chemicals on the eye is much like that of extreme heat, and these injuries are commonly designated as "burns." In the arts and manufactures, as well as occasionally in the household, sulphuric acid and nitric acid in concentration are used, while unslaked lime, mortar, and plaster, all of them more or less powerful alkalies, are in daily use in the construction of buildings.

The symptoms and clinical history of burns with acid are those of deep scalds.

The fluids in the conjunctival sac, which generally enter the eye in small quantity only, dilute the acid, so that the intensely destructive action of the acid is generally limited to the lids and the brow.

Concentrated crude sulphuric acid or oil of vitriol is occasionally thrown into the face with criminal intent, and may destroy the eye or produce extensive destruction of the soft parts, with great disfigurement. The treatment is that of severe burns of the eye. In fresh cases we may attempt to neutralize the acid by instilling alkaline solutions, such as sodium bicarbonate, borax, bicarbonate of potassium; lime water or milk may be employed.

In some cases explosion of the vessel containing acid takes place, and we have a complication of the corrosion by injury from splinters of glass, etc.

In injuries to the eye by burn with alkalies we have to deal most frequently with lime-burn. Unslaked lime is exceedingly pernicious to the integrity of the eye. It tends to absorb water rapidly, evolving great heat in the process of hydration or slaking, and acting as a most powerful caustic on mucous membranes. Unlike an acid, its action is increased by coming in contact with water, so that the tears, which act as a partial safeguard in burns with acid, only increase the extent of injury in the case of lime. Fresh mortar or plaster has a similar action; old plaster or old slaked lime is much weaker, although sufficiently caustic to produce severe irritation. The lime particles are found on the inner surface of the lids and in the conjunctival sac, and, in addition to the usual treatment, we must see that every bit of lime is carefully removed. In fresh cases the lime may be removed by irrigating the surface of the globe with some solution which

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forms a soluble salt of calcium and dissolves the lime. For this purpose we may employ a strong solution of sugar.

General treatment should be sustaining and, if necessary, somewhat stimulant, as most severe burns or scalds of the eye produce a certain degree of general prostration and nervous shock. In most cases the prognosis is unfavorable. The surface of the cornea is usually converted into a dense white leucoma, and symblepharon is a frequent result.

After lightning-stroke various injuries of the eye may be observed. Superficial burns of lids and cornea, singeing of eyebrow and lashes, may be the only injury. Cataract is a not infrequent result, a condition that has been attributed to an electrolytic action similar to that which produces curdling of milk during a thunder-storm. It may, however, be due to an irido-chorioiditis from injury, and be a secondary condition.

The opacity may take the form of a stationary posterior polar cataract, or the lens may become opaque and be extracted by the usual methods after maturation. Rupture and hemorrhage of the chorioid and retina, detachment of retina, optic atrophy, and neuritis have also been observed, while paralysis of the ocular muscles, with limitation of motility, ptosis, mydriasis, and loss of accommodation, may also be mentioned.

Hess[1] has directed the sparks of a large Leyden jar upon the ocular region of rabbits and cats, and has observed in these animals the gradual development of cataract. He argues from his experiments that the formation of lenticular opacities from a stroke of lightning is due in the first place to necrosis of the capsular cells, and not, as has been maintained by others, to coagulation consequent upon catalytic or thermic action.

Acute conjunctivitis is at times produced by the action of intense light, especially the electric arc light. Under appropriate treatment the inflammatory symptoms disappear in a few days.

Retinitis as the result of the action of light has been reported in several instances. It has occurred in persons who followed the different phases of a solar eclipse without protecting their eyes sufficiently. Charles A. Oliver[2] has published a clinical study of a case of double chorio-retinitis in the macular region following a flash of lightning and a flash of burning lycopodium. The day after the accident the patient noticed in the point of fixation a very bright spot surrounded by a rapidly moving grayish zone. At first this scotoma appeared in the right eye, but eleven days later it was also seen in the left eye. The scotomata disappeared in about four months, but central vision of the more intensely affected right eye was still much impaired at that time. Eighteen months after the accident vision was almost normal in both eyes. The ophthalmoscopic changes in the right eye-ground, drawn at a time when the scotoma was still present, are shown in

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[1] Carl Hess, Experimentelles über Blitzcataract, Internationaler Ophthalmologen-Congress, Heidelberg, 1888.

[2] Transactions of the American Ophthalmological Society, 1896.

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the appended picture from a sketch by Miss Washington, which I owe to the kindness of Dr. Oliver.

In the macular region the retinal tissue seemed to be slightly puffed into an irregularly flattened mass. The tissue glistened in places as though it were composed of transparent jelly. In an oblique direction the patch measured twice the diameter of the papilla, and was encircled by a narrow silvery band. At a later period only the faintest traces of these changes could be seen.

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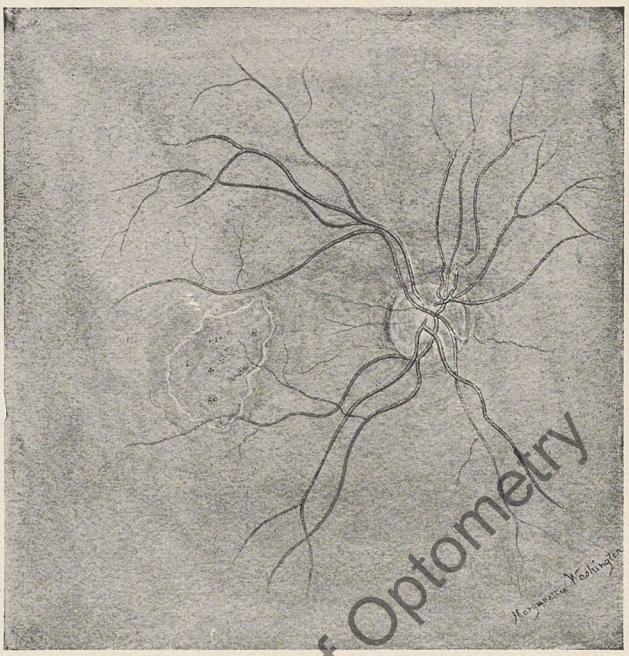
FIG. 15.



Macular chorio-retinitis following a flash of lightning. (Oliver.)

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SYMPATHETIC OPHTHALMIA.

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HISTORICAL.

According to Brondeau,[1] the first mention of the possibility of disease passing from one eye to the other is to be found in a work of Thomas Bartholinus:[2] "Yesterday I saw the daughter of a certain official in Cimbria, whose right eye had been blinded by a wound from a knife. The eye itself got well, but without sight, and the left eye, which was formerly a healthy eye, became the seat of incipient cataract." This observation was recorded in the latter part of the seventeenth century.

The second observation was made by Bidloo, 1649–1713, and is quoted by Jobert[3] in his work "Plaies par armes à feu." A long and irregular splinter of wood had pierced the eye, and, it being impossible to extract the splinter without opening the eyeball, the former was cut off close to the border of the cornea, and the rest of it was allowed to remain in the eye, in the hope that it would be thrown out by suppuration. The surgeon, however, was cruelly deceived, for the inflammation became violent and communicated itself to the other eye, and it was only with the greatest difficulty that the latter could be saved.

In 1741 Le Dran[4] speaks in a way which leaves no doubt that he recognizes the existence of sympathetic inflammation: "If the ordinary methods of subduing the inflammation fail, an abscess may have formed in the eyeball; if this be the case, it is necessary to split the eye open from one side to the other to let out the pus,—that is, where there is every evidence of the existence of pus. One recognizes this condition principally by the swelling of the lids and by the shooting pains which the patient feels. If, as in the case of abscesses elsewhere in the body, one waits until the pus forms, the patient can lose the sight by the inflammation communicating itself to the good eye along the length of the optic nerve."

[1] Brondeau, Des affections sympathiques de l'un des yeux à la suite d'une blessure de l'autre œil, Paris, 1858.

[2] Bartholinus, Bibliotheca Medico-practica, vol. iii.

[3] Jos. Jobert, Plaies par armes à feu, Paris, 1833.

[4] Le Dran, Traité ou réflexions tirées de la pratique sur les plaies d'armes à feu, Paris, 1737.

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Later (1802) Beer, according to Pagenstecher,[1] made the observation that where an inflammation has persisted in an eye for many years, and where sight is gone, heightened irritability and asthenopia make their appearance in the other eye. He advises that as long as the inflammation persists in the blind eye the relatively good eye should be the object of the greatest care and should be spared as much as possible, and he points to the possibility of the disease which is in progress in the blind eye causing blindness in the other eye.

In 1818 Demours[2] reports three cases in which he establishes the existence of sympathetic blindness. One was a case of a healthy young girl, who was watching a shoemaker at work. The latter, in making a sudden motion, struck the child in the eye with the end of his knife. The result was a violent and obstinate inflammation, and after several months the pupil of the eye was occluded. As to the other eye, the cornea remained clear, but the lens was opaque and the pupil was contracted, and the iris at this point was pressed forward. The little opening was just large enough for her to see objects about her. The second case was that of a woman who was shot in the right eye. The left eye remained good for a year after the accident, when sympathetic disturbance showed itself and ended in total blindness. The third case was that of a young man who was under observation for several years, in whom the healthy eye was the seat of constant attacks of sympathetic disease, and Demours says that the eye was preserved only by the most active measures. He closes the subject by calling attention to the danger which always menaces the sound eye in such cases, and emphasizes the necessity of watching carefully this eye. He speaks of the disease as one of the gravest in practice, and possibly beyond the resources of science. Demours was the first physician in France who called attention to this disease.

In von Ammon's[3] (1835) prize essay I find the following passage: "Traumatic iritis in an eye which has been wounded not infrequently passes over to the healthy eye. I have several times observed this diseased sympathy in eyes. The first was a case of an old man who suffered a wide, gaping wound of the sclera; the iris was prolapsed and much of it lacerated. Scarcely any iritis followed, but two months later uveitis showed itself in the other eye. The other was the case of a girl who was wounded in the right eye by the sudden explosion of a machine. Incurable blindness followed, and about four months later a wide-spread and rapidly progressing uveitis developed in the other eye." It is clear that while the physician is treating the wounded eye he should ever have in mind the condition of the sound eye, lest it also be attacked. The history of the disease, then, goes back to a period nearly one hundred and fifty

[1] Pagenstecher, Klinische Beobachtungen aus der Augenheilanstalt zu Wiesbaden, Heft 2, 1862, S. 47.

[2] Demours, Traité des maladies des yeux, Paris, 1818, t i. p. 360.

[3] Von Ammon und Walther's Journal, Neue Folge, Bd. i.

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years before Mackenzie, and, while the value of the earliest observations may be questionable, there can be no doubt that the disease was recognized by Demours as early as 1818.

Sympathetic ophthalmia was first described exhaustively by Mackenzie[1] in 1844. He was the first to describe it as a distinct disease and one fraught with great danger. "Whenever I see sympathetic ophthalmitis, even in the first stage, I know that I have to contend with an affection which, however slight its present symptoms may be, is one of the most dangerous inflammations to which the organ of vision is exposed."

In 1849 Tavignot,[2] speaking of sympathetic iritis of one eye following a wound of the other eye, dissents in some points from the views advanced by Mackenzie as to the symptomatology of the disease, and holds that the inflammatory phenomena are to be attributed to the wound of the ciliary body. He regards the disease as one in itself, but does not lay much stress upon its exceptional gravity, speaking of it as simply a sympathetic ciliary neuralgia which produces at first congestion and then inflammation.

In 1852 White Cooper[3] mentions the frequency with which he has met with sympathetic inflammation of one eye following a wound of the other. He speaks of the hopelessness of any treatment, and questions whether it would not be wise to enucleate the injured eye under such circumstances.

Two years later Prichard[4] reported twenty cases of sympathetic ophthalmia, and advised the enucleation of the injured eye so soon as the healthy eye showed signs of a serious inflammation.

In 1855 R. Taylor[3] reported eight cases. He expressed himself as opposed to extirpation of the eyeball, and thought that cutting away the cornea was all that was necessary.

In 1863 Critchett,[5] at a meeting in Heidelberg, spoke of the disease in these words: "That which especially claims interest in these cases is the tediousness, the insidiousness, and the obstinacy of the inflammation, the destructive influence which it exercises on vision, and the resistance it shows to all kinds of treatment." Von Graefe, in the discussion that followed, mentions, among other etiological factors, repeatedly recurring intra-ocular hemorrhage with sudden change in tension, also chalky deposits within the eye, which keep up a condition of sensitiveness. In 1866 von Graefe[6] says he is no longer of the opinion that simple changes in tension or recurrent intra-ocular hemorrhages can bring about an attack of sym-

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[1] W. Mackenzie, Practical Treatise on the Diseases of the Eye, London, 1854.

[2] Tavignot, Gazette des Hôpitaux, 1849.

[3] Moorem, Ueber sympathische Gesichtsstörungen, Berlin, 1869.

[4] Prichard, Journal of the British Medical Association, October, 1854.

[5] Critchett, Ueber sympathische Ophthalmie, Vortrag und Discussion, Zehender's Monatsblätter, Bd. i., 1863, S. 440.

[6] Von Graefe, Zur Lehre von der sympathischen Ophthalmie, Archiv f. Ophthal., xii., 2, 1866, S. 149.

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pathetic ophthalmia, but that these conditions are simply the introductory phenomena.

DEFINITION.

Sympathetic ophthalmia may be defined as an inflammation which is usually plastic in character, but sometimes serous, which affects the iris, ciliary region, and chorioid of one eye, and which has its origin in a traumatic inflammation of the same parts in the other eye. Two factors, then, enter into the composition of this disease: first, a traumatic inflammation of one eye; second, a plastic uveitis of the other eye; and to these may be added a third factor, time, for a limited period of time always elapses before the outbreak of the sympathetic disease. These are the three fundamental elements of true sympathetic ophthalmia: the absence of one of these elements should render the diagnosis doubtful, and the existence of all three would hardly warrant any other interpretation.

ETIOLOGY.

Injuries undoubtedly play the most important part in the causation of sympathetic ophthalmia. The kinds of injuries are usually penetrating and lacerating wounds from sharp instruments, such as scissors and knives, and the injuries resulting from the entrance and lodgement in the eyeball of small splinters, fragments of a percussion-cap, particles of stone, glass, and chips of iron. A mere blow on the eyeball without causing any rupture, according to Mackenzie,[1] has been known to affect the other eye sympathetically. Mackenzie[1] described a case in which sulphuric acid was thrown into the left eye of a girl. The consequence was destruction of the cornea and union of the whole of the upper eyelid to the remains of the eyeball. Two months later she began working in a cotton-mill. This brought on a severe attack of sympathetic ophthalmia of the other eye, ending in haziness of the cornea, discoloration of the iris, immobility of the pupil, and great deterioration of vision. This illustrates the diversity of injuries which may give rise to sympathetic ophthalmia. Subconjunctival rupture alone of the eyeball has given rise to sympathetic disease, and Schirmer[2] has collected a number of cases of this kind. I think, however, that many of the cases are open to grave criticism, an opinion which it is evident that Schirmer himself shares. The following case is reported by Gunn.[3] Three weeks before the patient came under observation an accident had befallen the left eye, by which the lens had been forced out under the conjunctiva. Twenty days later, or about the time he was first seen, plastic iritis showed itself in the other eye. The injured eye was enucleated, and the sympathizing eye got well. An anatomical examination

[1] W. Mackenzie, loc. cit.

[2] O. Schirmer, Klinische und pathologisch-anatomische Studien zu Pathogenese der sympathischen Augenentzündung, Archiv f. Ophthal., xxxviii., iv., S. 95, 297.

[3] Gunn, On Sympathetic Inflammation of the Eyeball, Royal London Oph. Hosp. Rep., xi., 1886.

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of the enucleated eye showed plastic uveitis. Knapp[1] reports a case of sympathetic ophthalmia following traumatic dislocation of the iris under the unbroken conjunctiva. These two seem to be genuine cases; and the same may be said of a case reported by Sachs,[2] of one by Alt,[3] and finally of one by Deutschmann.[4] Other cases of a similar character, but of doubtful value in this connection, have been reported by Mooren,[5] Argyll-Robertson,[6] Bresgen,[7] Pagenstecher,[8] Savary,[9] Ayres,[10] [11], Manolescu,[12] and Arlt.[13]

Penetrating wounds are, as a rule, the kind of injuries which give rise to this disease. Such wounds are nearly always associated with loss of aqueous or vitreous humor and with intra-ocular hemorrhages. A very common and dangerous site for an injury is the ciliary region. This is really the seat of life, so to speak, of the eye, and it makes no difference whether the wound be large or small, it is always full of danger. Mooren[14] says that the common idea that large wounds less often give rise to sympathetic ophthalmia than small ones is not confirmed by his experience, for he often saw the disease follow the entrance of small particles of iron, and often saw it result from the bursting of an eye by a blow with a stick. Traumatic cataract in such cases is to be regarded as an unfortunate complication. So soon as the lens-capsule is ruptured the lens swells up and presses against the wounded iris and ciliary body, and acts as a constant irritant. In connection with wounds of the ciliary region, Mooren calls attention to the comparatively short time it takes for wounds of the sclera to heal when these wounds are located behind the ciliary region. He reports a case where the inner equatorial part of the sclera was torn by a powerful blow with a flail, and where a very large part of the corpus vitreum had escaped, giving the eye the appearance of being squeezed in, and yet recovery followed in a comparatively short time.

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[1] Knapp, Transactions of the American Ophthalmological Society, vol. vi. p. 513.

[2] Sachs, Ueber traumatische Scleralrupturen im vorderen Bulbusabschnitt, Archiv für Augenheilk., xx., 1889.

[3] Alt, Studien ueber die anatomischen Gründe und das Wesen der sympathischen Ophthalmie, Archiv für Augenheilk., vi., 1877.

[4] Deutschmann, Ueber die Ophthalmia Migratoria, Hamburg und Leipzig, 1889.

[5] Mooren, Fünf Lustren ophthalmologischer Wirksamkeit, No. 155, Wiesbaden, 1882.

[6] Argyll-Robertson, Case of Sympathetic Neuro-Retinitis Pigmentosa, Royal London Ophthal. Hosp. Reports, vii., 1871.

[7] H. Bresgen, Fälle vom sympathischen Erkranken des Auges, Wiener medizinische Wochenschrift, Nr. 45 u. 46, 1878.

[8] H. Pagenstecher, Zur Casuistik der Augenverletzungen, Archiv für Augenheilk., viii., 1879.

[9] Savary, Nouvelle observation à joindre au dossier des ophthalmies sympathiques, Ann. d'Oculist., t. lxxvi., 1876.

[10] Ayres, Sympathetic Inflammation, Archives of Ophthalmology, xii., 1883.

[11] Ayres, Five Cases of Sympathetic Ophthalmia, Archives of Ophthalmology, vii., 1878.

[12] Manolescu, Aniridie et Aphakie traumatiques, Archives d'Opht., v., 1885.

[13] Arlt, Klinische Darstellung der Krankheiten des Auges, 1881, S. 213.

[14] Mooren, Ueber sympathische Gesichtsstörungen, Berlin, 1869.

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Mackenzie[1] thinks that the disease is more apt to be excited if the wound has produced a protrusion of the iris and such a cicatrix of the cornea and sclera as keeps the portion of the iris which had not protruded perpetually on the stretch. An unsuccessful cataract operation, then, can bring about sympathetic ophthalmia. When the retina has been wounded I think the gravity of the injury is greater. Wounds which pass through the cornea and the pupillary border of the iris, even when a traumatic cataract results, are not so apt to give rise to sympathetic disease as wounds in which the ciliary border of the iris has been involved. Traumatic cataract of itself has no significance in the causation of the disease. It is only when the swollen lens presses upon an injured and inflamed ciliary body that the former can be regarded as a causal factor. Among other causes may be mentioned the operations of iridodesis, discission, iridectomy, and reclination. Mackenzie makes the statement that he has never seen the disease follow any of the operations for cataract, and yet since Mackenzie's day there have been reported at least a hundred cases of sympathetic ophthalmia following the extraction of cataract.

Most text-books mention intra-ocular tumors as among the causes of sympathetic ophthalmia. Schirmer[2] has collected the reports of thirty such cases; in twenty-eight the tumor was supposed to be a melano-sarcoma, and the other two were gliomas. In nine of these cases sympathetic irritation, and not inflammation, was present. The histories of five cases were too imperfect to allow any decided opinion. In three of the cases in which a genuine inflammation existed in the sound eye there was no tumor, and in one case it was doubtful whether the primarily affected eye was the seat of a tumor or of a chronic inflammation. In six cases the eyeball ruptured before the outbreak of the sympathetic disturbance; so that it is impossible to say whether the latter was due to the tumor or to infection from without the eye. In only three cases (two reported by Milles[3] and one by Deutschmann[4]) was there a strong probability that the sympathetic inflammation was attributable to the existence of a tumor. In all these cases irido-cyclitis was present, and Schirmer concludes that the real agent is the irido-cyclitis, and that a tumor of the uveal tract is capable of exciting sympathetic inflammation only when associated with irido-cyclitis.

From this survey of the facts I am disposed to regard the chorioidal sarcomas as very doubtful agents in the production of sympathetic ophthalmia.

Cysticercus is also mentioned in most text-books as one of the causes of sympathetic ophthalmia, but so far as I can find there have been reported in this connection only two cases of cysticercus, one by Alfred Graefe,[5] and the other by Jacobson,[6] and both were instances of sympathetic am-

[1] W. Mackenzie, loc. cit. [2] O. Schirmer, loc. cit.
[3] Milles, Curator's Pathological Report, Royal London Oph. Hosp. Rep , xi., 1887.
[4] Deutschmann, loc. cit. [5] Graefe's Archiv für Ophthalmologie, xli., S. 123.
[6] Jacobson, Zwei Fälle von intraocularem Cysticercus mit Sectionsbefund, Archiv für Ophthalmologie, xi. 2, 1865.

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blyopia, and cannot possibly be regarded in the light of true sympathetic inflammation.

The following interesting communication is made by F. Pincus.[1] Man, forty-two years of age. The right eye always has been sound, but the left eye for the past thirteen years has been the seat of violent inflammation, —in fact, was never free from inflammation. When first seen there was dislocation of the lens in the left eye. The luxated lens was extracted. Three weeks later the patient, contrary to advice, departed for home, and a month afterwards returned with his left eye swollen and sensitive to touch and the anterior chamber filled with a yellowish exudate, while in the right eye there was plastic irido-chorioiditis. The left eye was enucleated, and in three months the other eye was entirely well. The anatomical examination revealed a subretinal cysticercus. The scar from the operation was imperfectly healed, and between the lips of the wound there was a mass of infiltrated tissue, and at this point micro-organisms were found. It is evident, however, that the cysticercus had nothing to do with the causation of the sympathetic affection, but that the latter can be traced to the operation for the luxated lens. It is remarkable that sympathetic irritation did not appear in the right eye, when we consider that the ciliary nerves of the left eye had been in a condition of irritation for thirteen years.

As regards ossification within the eye as a cause of sympathetic disease, there are good reasons for believing that the ossification is not directly responsible, but that the outbreak of a sympathetic inflammation in such cases is to be attributed to a coexisting inflammation in the sympathizing eye, which inflammation does not depend upon the presence of the ossification. It is certainly a fact that in the majority of cases where ossification existed sympathetic ophthalmia was absent, and that under the microscope no fresh inflammatory changes could be detected. In this connection cases have been reported by Knapp[2] and Schirmer.[3]

Cases of sympathetic ophthalmia have been known to result from herpes zoster ophthalmicus. Such cases have been reported by Noyes,[4] Jeffries,[5] and Guérin.[6] Lebrun[7] reports a case of sympathetic ophthalmia following the bite of a leech which had been ordered by a physician. Panas[8] relates a case in which sympathetic disease followed upon tattooing the cornea.

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[1] Pincus, Anatomischer Befund von zwei sympathisirenden Augen darunter eins mit Cysticercus intraocularis, Archiv für Ophthalmologie, xl.

[2] Knapp, Ueber Knochenbildung im Auge, Archiv f. Augenheilkunde, ii., 1871.

[3] O. Schirmer, loc. cit.

[4] Noyes, Herpes Zoster Ophthalmicus causing Loss of the Corresponding Eye and Subsequent Loss of the Opposite Eye, Trans. Amer. Oph. Soc., 1873.

[5] Jeffries, Two Cases of Herpes Zoster Ophthalmicus destroying the Eye, Trans. Amer. Oph. Soc., 1873.

[6] Guérin, Du zone ophthalmique, Thèse de Paris, 1884.

[7] Lebrun, Ann. d'Oculist., t. lxiv. p. 136.

[8] Panas, Gazette des Hôpitaux, No. 85.

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Agnew,[1] Webster,[2] Mathewson,[3] Peck,[4] Rossander,[5] and Mooren[6] report cases of sympathetic ophthalmia resulting from symblepharon, while there are several cases on record where the same affection followed the wearing of an artificial eye.

In summing up the various causes of sympathetic ophthalmia, it may be emphasized that whatever can cause an irido-cyclitis in one eye can indirectly bring about sympathetic inflammation in the other.

SYMPTOMS.

The earliest symptom is the rapidity with which the accommodation becomes tired as soon as the slightest attempt is made to fix an object, whether large or small, far or near. This symptom is especially characteristic of that form of sympathetic disturbance known as sympathetic irritation. It is possible for these irritative symptoms to be lacking, and the first intimation that the patient has may be a mist around everything, a gradual suffusion of objects. In the case of small children, who are apt to overlook accommodative asthenopia, this last symptom may be the first striking one, and it always means positive tissue-changes and that the disease has made dangerous headway. In those cases of sympathetic ophthalmia which have come under my observation pain was conspicuously absent. The onset of the disease was generally insidious, and blindness followed with practically no suffering whatever. Generally every effort made by the patient to see fine objects is attended by increased secretion of tears and redness of the conjunctiva, and if the effort is persisted in headache follows, with increased congestion of the primarily affected eye. As time goes on, the outlines of objects become hazy and there is a falling off in central vision. Suddenly the pericorneal injection becomes more intense, and that, too, without there being any special subjective symptoms present to account for it. Owing to a slight cloudiness in the media, the optic nerve looks indistinct and generally red, though there may be actual papillitis present, such cases having been seen by Alt,[7] Spalding,[8] and others in the milder form of sympathetic ophthalmia.

The tension in sympathetic ophthalmia is variable; the earliest stages are associated with very slight increase of tension, followed by vacillating conditions, mounting up to a high grade in the glaucomatous stage, while the intra-ocular tension at the last is decidedly diminished, the eyeball feeling

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[1] Agnew, quoted by Deutschmann, op. cit., p. 89.

[2] Webster, Sympathetic Neuro-Retinitis, New York Med. Rec., 1881.

[3] Mathewson, quoted by Deutschmann, op. cit., p. 89.

[4] Peck, A Case of Sympathetic Ophthalmia due to Symblepharon, New York Med. Rec., 1881.

[5] Rossander, Contributions à l'étude des ophthalmies sympathiques, ref. in Ann. d'Oculist., t. lxxv., 1876, p. 301.

[6] Mooren, Fünf Lustren ophthalmologischer Wirksamkeit, No. 155, Wiesbaden, 1882.

[7] Alt, Archives of Ophthalmology and Otology, vol. v., 1876, p. 395.

[8] Spalding, Trans. Amer. Oph. Soc., 1883, p. 486.

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soft to the touch. The aqueous humor becomes clouded and the iris hyperæmic. This latter condition gives a greenish hue to a blue iris and a brownish shading to dark eyes. These changes are generally attended with little or absolutely no pain. Pagenstecher[1] has called attention to the fact that this kind of iritis differs from the ordinary iritis in that in the former variety the pupil can readily be dilated in spite of the synechiæ. The process has been known to come to a stand-still at this stage and never reappear, leaving the patient with comfortable vision. This, however, is very rare, and has been observed only in the milder form of sympathetic inflammation known as iritis serosa. As a rule, the process returns with renewed intensity, and at every attack the pupil is harder to dilate, the injection more intense, the hyperæmia of the iris more pronounced, the anterior chamber wider. Pain is variable: in some cases it is very slight, and in others it is intense. There is always more or less torpor of the retina. Small grayish dots appear on the posterior surface of the cornea, and soon we have before us the picture of cyclitis. Synechiæ appear extending all around the pupil, and there result wide-spread circulatory changes in the deeper structures of the eye. Recession of the iris periphery is seen.

Mackenzie[2] has called attention to flexibility of the cornea, bogginess of the sclera (both of which point to degenerative changes in the retina, chorioid, and vitreous, the latter becoming fluid), and clouding of the lens-capsule as among the earlier symptoms.

Mooren[3] mentions a peculiarity of this kind of inflammation in its late stages which distinguishes it from other forms of iritis,—namely, the great rigidity of the iris-tissue. He says it is exceedingly difficult to perform a successful iridectomy on these cases, it being almost impossible to get a wide pupillary opening owing to this rigidity. When a portion of the iris is drawn out it shows no tendency to return to its former position when set free, but remains lying in the wound, serving to heighten the intensity of the inflammation and bring about the complete closure of the pupil with a thick exudate. This condition is the rule in the sympathetic form of iritis. Mooren calls it a kind of felting which changes the entire uveal tract—at least the iris and the ciliary body—into a rigid tissue. The further changes von Graefe attributes to proliferative processes on the posterior surface of the iris which have the effect of obstructing the circulation in the ciliary region. This proliferative process frequently brings about such nutritive changes that phthisis bulbi follows. Again, it develops with such rapidity and vigor that the iris is forced against the posterior surface of the cornea, increased intraocular tension follows, and there are all the symptoms of glaucoma. This condition is always attended with the most violent ciliary neuralgia, and it is remarkable that the pain in these cases is far more intense in the sympa-

[1] Pagenstecher, quoted by Mooren, Ueber sympathische Gesichtsstörungen, Berlin, 1869.

[2] W. Mackenzie, loc. cit.

[3] Mooren, Ueber sympathische Gesichtsstörungen, Berlin, 1869.

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thizing eye than it ever was in the primarily affected eye. It is quite certain that in most cases the primarily affected eye is blind before the outbreak of sympathetic inflammation. There are cases, however, recorded in which vision was still present at the time of the outbreak of the sympathetic affection; and I once met with such a case.

Hirschberg[1] is of the opinion that the fundus of the eye undergoes peculiar and characteristic changes in sympathetic ophthalmia. These changes somewhat resemble those seen in syphilis, and consist in the occurrence of small, round, white spots in the periphery. These spots lie generally next to the branches of the retinal blood-vessels, and sometimes just behind them. Hirschberg's observations extend over only two cases, and until similar observations have been repeatedly made it cannot be said that the changes just described are those peculiar to sympathetic ophthalmia. While the disease is in progress it is usually impossible to obtain a satisfactory view of the fundus, owing to the cloudiness of the media; and it is only in those very rare cases of recovery when this cloudiness has passed away that we can get any idea as to the character of the retinal and chorioidal changes.

Mackenzie[2] has called attention to the frequency with which the following constitutional symptoms arise in the course of sympathetic ophthalmia: quickness of the pulse, thirst, marked buffy coat on the blood drawn from a vein, a pallid complexion, and obstinate constipation. A degree of ill health, in fact, has generally resulted from the confinement, want of exercise, and medical treatment necessary for the cure of the original accident, and in this debilitated state the patient is attacked by the sympathetic disease.

SYMPATHETIC IRITIS SEROSA.

There is no doubt that this form of sympathetic inflammation is fraught with far less danger to the eye than is the plastic irido-cyclitis. The symptoms are the usual ones of iritis serosa. There is a hypersecretion of the aqueous humor, which in addition becomes very cloudy, and deposits of various sizes and shapes are seen on the surface of the cornea and on the anterior capsule of the lens. Pericorneal injection is not particularly pronounced; the anterior chamber is generally deeper than normal, intra-ocular tension is usually increased, the pupil is somewhat smaller than normal, and the iris reacts but feebly. Pain is not often present in the earlier stages of the disease, and is hardly ever a marked symptom. Sometimes small particles may be seen floating about in the anterior chamber. As I have just remarked, this affection is not so dangerous as the plastic irido-cyclitis, and Donders and von Graefe have sought to explain this by assuming that iritis serosa has not the property of changing into the more pernicious form of sympathetic inflammation. Mooren[3] reports the following case. "A

[1] Hirschberg, Centralblatt für praktische Augenheilkunde, 1895, S. 80.

[2] W. Mackenzie, loco citato.

[3] Mooren, Ueber sympathische Gesichtsstörungen, Berlin, 1869.

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peasant woman came to me with a prolapsed iris and cyclitis in one eye and iritis serosa in the other eye. Enucleation of the primarily affected eye was performed, and two days later, as most of the injection had disappeared from the second eye, an iridectomy was performed. The excised portion of the iris showed a peculiar rigidity of the tissue. The eye healed in the usual time, and the woman returned home. I had supposed the patient permanently well, when some weeks later she returned with a fresh inflammation. This time the iritis was of a violent type, and there was also present a purulent deposit on the posterior surface of the cornea. In spite of all medication the hypopyon increased; the pain became unbearable and extended entirely over that side of the head. The ciliary body was very sensitive to the touch, and the eyeball took on a glaucomatous hardness. Had the patient presented herself to me in the first instance with these symptoms, I should have designated it as a case of glaucoma complicated with cyclitis. The process ended in phthisis bulbi."

On January 3, 1893, a girl of twelve years came to the clinic of the Presbyterian Eye and Ear Hospital, Baltimore. She had run the point of a pair of scissors into her left eye six weeks before. There was a horizontal wound through the cornea and sclera, and a portion of the iris had been incarcerated. The pupil was completely closed up, and the iris puckered and thrown into folds. Tension minus, and light-perception doubtful. The interesting point, however, was the condition of the right eye. Five weeks after the injury the sight in the right eye began to grow dim, and when she came to the hospital she was suffering with iritis serosa in this eye. The aqueous humor was faintly clouded, and there were three posterior synechiæ. The vision was about one-half. Enucleation was advised, and the father consented. The eye was removed, and atropine was instilled every four hours into the other eye. The adhesions yielded, the aqueous humor cleared up, and recovery was perfect in three weeks. That was nearly a year ago, and there is no reason to believe that any relapse has occurred. In these cases, even when vision has been markedly reduced, recovery has taken place without even an operation on the exciting eye, such cases being reported by Mills and Frost[1] and Noyes.[2]

Iritis serosa, then, may be regarded as a comparatively benign form of sympathetic inflammation, though it does sometimes pass over into the pernicious form, plastic irido-cyclitis.

SYMPATHETIC PAPILLO-RETINITIS.

In a certain number of cases it is evident that the irido-cyclitis is associated with a papillitis, and it seems difficult to explain the exact relation that the two affections bear to each other. Doubtless papillitis exists in many cases of sympathetic ophthalmia, and that, too, as a primary affection, and owing to the cloudiness of the media it is impossible to make out this

[1] Mills and Frost, Trans. Oph. Soc. United Kingdom, vol. iii., 1883, pp. 63 and 73.
[2] Noyes, Diseases of the Eye, p. 490, 1890.

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condition. On the other hand, there are not a few cases reported where there was no doubt as to the existence of a papillo-retinitis without any implication of the uveal tract. Such cases are reported by Hirschberg,[1] Pflüger,[2] Pooley,[3] Brailey,[4] Gepner,[5] Alt,[6] Eversbusch,[7] and Spalding.[8]

The observation of Spalding[8] is of special interest. A woman sixty-five years old was struck in the right eye by a cow's horn. Violent pain and blindness followed. Thirty-five days later the vision in the left eye began to decline, so that it was not long before the patient had to be led about. Spalding first saw her eleven weeks after the injury. She could count fingers at four inches. There was typical neuro-retinitis. The media were all absolutely clear. The capsule of the lens showed no evidence of previously existing inflammation. The injured eye was immediately enucleated, and without the slightest medication the vision in the sympathizing eye in the course of a few months had risen to two-thirds. The chorioid had remained normal.

This variety of sympathetic ophthalmia, in contradistinction to that form with which we generally meet, shows no tendency to relapses.

Schirmer[9] calls attention to the fact that the disease has never been observed after the enucleation of the injured eye. It is a benign affection, and a restoration to normal vision is the rule. The papillitis seems to be absolutely dependent upon the injured eye. All these facts, Schirmer thinks, would warrant the conclusion that the disease is not due to the migration of bacteria from the injured eye, but that the metabolic products of the organisms reach the second eye and give rise to the papillitis. This explanation was suggested by Deutschmann.[10] Of course the optic nerves constitute the route followed by the metabolic products, and this has been proved possible by the experiments of Horner and Knies,[11] who found that injections of fluorescin and other chemical agents into the subpial space of the optic nerve at its peripheral end were followed by the appearance of these agents all along the optic nerve and in the other eye.

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[1] Hirschberg, Klinische Beobachtungen, p. 35, Wien, 1874.

[2] Pflüger, Zur sympathischen Ophthalmie, Correspondenz-Blatt für Schweizer Aerzte, Nr. 7 u. 8, 1875.

[3] T. R. Pooley, A Case of Sympathetic Neuro-Retinitis, American Journal of Ophthalmology, 1884.

[4] Brailey, Sympathetic Neuro-Retinitis, Trans. Oph. Soc. United Kingdom, 1884.

[5] Gepner, Eine seltene Art von sympathischer Augenaffection, Centralblatt f. praktische Augenheilk., 1886.

[6] Alt, A Case of Sympathetic Neuro-Retinitis, American Journal of Ophthalmology, 1884.

[7] Eversbusch, Bericht über 1420 in der Münchner Augenklinik ausgeführte Staarentbindungen, Archiv für Augenheilkunde, xiii., 1884; Sympathische Augenentzündungen, Mittheilungen a. d. kgl. Universitäts-Augenklinik zu München, Bd. i., 1882.

[8] Spalding, loc. cit.

[9] O. Schirmer, loc. cit.

[10] Deutschmann, Ueber die Ophthalmia Migratoria, Hamburg und Leipzig, 1889.

[11] Horner und Knies, Ueber Opticusinjectionen, Verhandlungen der Heidelberger Oph. Ges., 1882.

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DIAGNOSIS.

With respect to the diagnosis, it would be well to call to mind the points which I have laid down as the fundamental factors of the disease. It must be remembered, however, that this disease, as Schweigger has said, does not have its own peculiar train of symptoms by which it can be invariably and immediately recognized. We have a right to regard the case as one of genuine sympathetic ophthalmia if we find well-defined objective symptoms of a plastic irido-cyclitis in an eye which had remained sound for three weeks after the fellow-eye had been the seat of a traumatic inflammation. It is the opinion of many that when the disease breaks out after a long interval,—that is, after the injured eye has become atrophic,—fresh inflammatory processes have sprung up in the atrophic eye. It is, of course, necessary to demonstrate the existence of this fresh inflammation. The point that so many make, that in such atrophic eyes the upper half of the ciliary region is very sensitive to pressure, is, I think, of very doubtful value. A great many, if not all, injured eyes which have become atrophic are sensitive to the touch at this point in the ciliary region, and they remain so for a long time without ever giving rise to sympathetic ophthalmia. In sympathetic irritation, on the other hand, we have a more positive characteristic,—namely, the immediate disappearance of all symptoms of irritation with the enucleation of the primarily affected eye. If these symptoms of irritation fail to disappear immediately the sympathetic connection is rendered somewhat doubtful, and if they continue after the enucleation we cannot look upon it as a genuine case.

COURSE.

The course of the disease is always tedious. After many exacerbations the disease can come to a point where it appears to stop, and here it may remain for many years and the patient have enough vision to get about. Usually, however, the pupil is closed with organized exudates which cut off the communication between the two chambers of the eye; in addition to this, the posterior surface of the iris becomes glued down to the anterior capsule of the lens, and as a consequence of these conditions the nutrition of the uveal tract is so disturbed that atrophy of the ball results. Some observers assert that cases of sympathetic irritation can pass over into genuine sympathetic inflammation, but I find no such cases on record. According to Schweigger,[1] this has not been proved. It is certainly true that the symptoms of irritation can be present for a long time without passing over into inflammation, and sympathetic inflammation often makes its appearance without any previous symptoms of irritation.

COMPLICATIONS.

Cerebral symptoms have been reported in connection with an attack of sympathetic ophthalmia.

[1] Schweigger, Augenheilkunde, S. 337.

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Snellen[1] reports a case of a man who was injured in the eye while opening a bottle; three weeks later there was present purulent infiltration of the cornea; the cornea was incised and the lens let out. A few days after this—six weeks after the injury—sympathetic cyclitis broke out in the other eye, accompanied by marked disturbances in hearing and violent headache and delirium. The injured eye was enucleated, and improvement followed slowly, but the cerebral symptoms disappeared. The patient, however, remained deaf and blind. The sympathizing eye became atrophic.

Risley,[2] Galezowski,[3] and Deutschmann[4] report somewhat similar cases, but there is not sufficient reason for believing that meningitis was present in any of these cases, and it certainly cannot be looked upon as a possible complication in the course of sympathetic ophthalmia.

Mackenzie[5] says sympathetic ophthalmitis may be complicated with scrofula and assume a good deal of the scrofulous character, or it may be complicated with syphilis. The history, however, will in general prevent any difficulty in the diagnosis.

FREQUENCY OF OCCURRENCE.

Sympathetic ophthalmia is unquestionably a rare disease; just how often it occurs it is impossible to say. It is unfortunate that the reports of the numerous large eye hospitals, not only in this country but abroad, are so arranged that they are nearly worthless as aids in developing any scientific subject. But were these hospital reports arranged with such an object in view, I am sure that with regard to sympathetic ophthalmia one would be struck by the rarity of its occurrence when one sees how often the conditions arise. Penetrating wounds of the ciliary region are almost every-day occurrences, and yet how very seldom do we see sympathetic ophthalmia follow! A connection of several years past as consulting surgeon with two large railroads, and also professional work for some time in a thickly populated mining district, have given me numerous opportunities of seeing this character of wounds, and only once under these circumstances have I seen sympathetic ophthalmia. It seems quite certain that the disease is less often seen nowadays than it was years ago, and I have no doubt that this may be explained by the better methods which now prevail in the treatment of injuries. I refer, of course, to methods of cleanliness and to antisepsis.

Ohlemann found in five hundred and fifty-six severe injuries that sympathetic ophthalmia occurred only twice.

It is evident, then, that the disease is of exceptional occurrence.

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[1] Snellen, Discussion on Sympathetic Ophthalmia, Trans. Internat. Med. Congress, London, 1881, vol. iii.

[2] Risley, A Case of Sympathetic Neuro-Retinitis with Consecutive Serous Iritis, Jour. Amer. Med. Assoc., iv., 1885.

[3] Galezowski, De l'épilepsie avec névrite optique, Recueil d'Opht., 1886.

[4] Deutschmann, Ueber die Ophthalmia Migratoria, Hamburg und Leipzig, 1889.

[5] W. Mackenzie, loc. cit.

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SYMPATHETIC IRRITATION.

This condition was once regarded as the inevitable forerunner of sympathetic inflammation, but it is needless to say that the latter makes its appearance sometimes without any symptoms of irritation, and, furthermore, it is well known that the condition of sympathetic irritation is a much more frequent affection than genuine sympathetic ophthalmia, that it often retains its peculiar clinical features for years, showing no tendency to pass over into inflammation, and that in several vital points these clinical features differ from those met with in sympathetic ophthalmia.

To Donders is due the honor of first separating the two affections. The condition shows itself in great photophobia, and frequently by pains in the head and orbit. Blepharospasm and lacrymation are always marked, with sometimes injection of the conjunctiva at the corneal margin. The aspect of the disease reminds us much of what we see in phlyctenular conjunctivitis. The pains usually occupy the upper half of the forehead and temples, and sometimes they extend to remote parts of the head. The neuralgia in some of these cases of sympathetic irritation is remarkable for its violence. It is not infrequently remittent in character. The photophobia is variable, though not, as a rule, so intense as that seen in the corneal inflammations. The field of vision sometimes suffers a concentric narrowing. There is an inability to see objects with distinctness. Shadows and clouds are frequently seen when an effort is made to look at an object.

Liebreich has called attention to another symptom with which he has not infrequently met,—that is, the disappearance of objects. Here the central vision is preserved, but a more or less complete obscuration of objects takes place from time to time: this obscuration lasts several seconds, or possibly a minute, and then the objects appear as distinct as ever. The disease is characterized by great prominence of subjective phenomena, with absence of structural lesions. The pupil is generally small, but the movements of the iris are intact. I have never seen any sluggishness nor paralysis of accommodation. Noyes[1] is of the opinion that the range of accommodation is diminished; such a condition, however, I have not seen.

Reich[2] reports a case of spasm of accommodation. Very rarely do these attacks show themselves with anything like periodicity, though the possibility of such an occurrence has been demonstrated, as, for example, in the following case, reported by Laqueur.[3] A man twenty years of age had lost his eye through a wound ten years before, and since that time had suffered with attacks of sympathetic irritation in the other eye. These attacks came on twice a year with great regularity in the spring and autumn. They persisted for three or four weeks, and prevented all work. There was not the slightest trace of inflammation.

I met with the following condition of sympathetic irritation in a miner

[1] Noyes, Diseases of the Eye, p. 490, 1890.
[2] Reich, Annales d'Oculistique, 1876, t. lxxv. p. 14.
[3] Laqueur, Étude sur les Affections sympathiques de l'Œil, Paris, 1869.

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who had lost one eye from a bullet wound many years before. Ever since the injury his other eye had once a year been the seat of attacks of sympathetic irritation lasting several days at a time. Between the attacks his good eye gave him absolutely no trouble. The removal of the injured and atrophic eyeball was followed by complete cessation of these attacks.

The following typical case of sympathetic irritation was observed by Donders.[1] A man was struck in the right eye with a piece of iron. Violent inflammation followed, with subsequent blindness a few weeks after the injury. While the eye was still painful the patient went to work again. Unfortunately, he was struck in the left eye with a particle of iron a few days later. The eye immediately became red, and lacrymation and photophobia were intense. This condition persisted for two years, the man believing that he was completely blind. He was tortured day and night with pain, and his face was actually deformed by reason of the intense photophobia. When Donders first saw the patient the right eye was atrophic, irritated, and painful. The left eye, which could be opened only with the greatest effort, showed a normal iris and cornea; the pupil was black and very much contracted. Under the supposition that it was a case of sympathetic irritation, the right eye was immediately enucleated. Two hours later the patient was able to open his eye well enough for Donders to establish the existence of normal vision. It appears that the existence of an insidious focus of inflammation in the first eye is sufficient to bring about an exaggeration of the reflex phenomena just as soon as the second receives the slightest injury. The weight of opinion is against the idea that this type of sympathetic disease can pass over into sympathetic inflammation,—an opinion which Donders himself shared. In this connection Mooren[2] mentions a case of sympathetic irritation associated with iritis; and while he does not say in so many words that the irritation changed into the iritis, he is of the opinion that the iritis influenced the obstinacy of the sympathetic irritation, for on the removal of the first eye the symptoms of irritation failed to disappear. In fact, the sympathetic irritation disappeared only after a wide iridectomy was performed. It seems to me, however, that the iritis in this case was simply a coincidence, and had nothing to do with the irritative symptoms.

Lawson,[3] Rossander,[4] and Mauthner[5] report cases in which sympathetic irritation has passed over into genuine inflammation; but all are capable of other interpretations; in fact, so far as I know, there are no well-established cases on record where such a transition has occurred.

The disease manifests itself at periods ranging from two and three months to fifteen and twenty years after the injury of the first eye. Cer-

[1] Donders, quoted by Mooren, Ueber sympathische Gesichtsstörungen, Berlin, 1869.
[2] Mooren, Ueber sympathische Gesichtsstörungen, Berlin, 1869.
[3] Lawson, Royal London Oph. Hosp. Rep., 1882, vol. x. p. 2.
[4] Rossander, loc. cit.
[5] Mauthner, Die sympathischen Augenleiden, Wiesbaden, 1881.

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tainly cases of sympathetic irritation are oftenest met with when the injured eye has been blind for a great length of time. This is in strong contrast to sympathetic inflammation, which usually breaks out more promptly. The chances are, then, that after the fifth month the irritative form of sympathetic ophthalmia will be the form most likely to occur. There seems to be absolutely no reason for believing that the disease is communicated to the sound eye otherwise than through the medium of the ciliary nerves. The following case of sympathetic irritation was reported by Greeff,[1] and is especially interesting on account of the physiology of the field of vision.

FIG. 1.

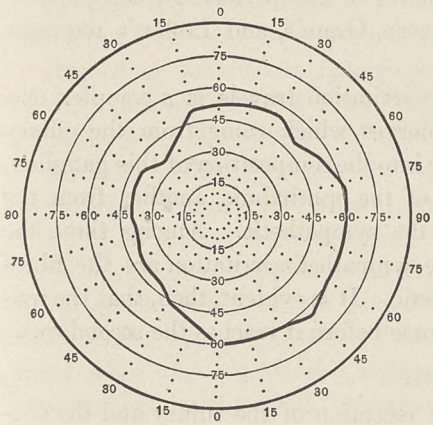


FIG. 2.

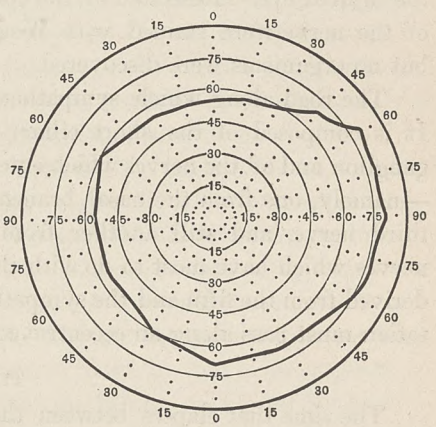
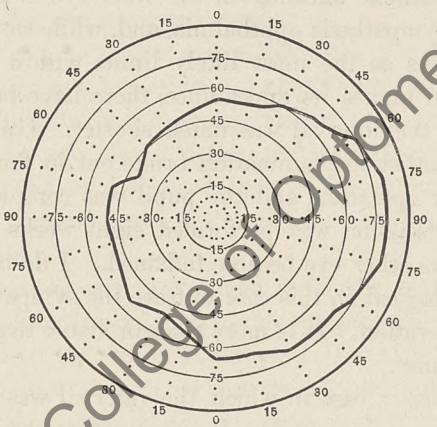


FIG. 3.



P. Grammann, sailor; wounded in the left eye on May 24, 1891, with a piece of steel. Nothing was removed from the eye. He was obliged to lie in bed with both eyes bound up for twenty days, using leeches and atropine. Immediately after the injury he was conscious of a pricking sensation in the region of the forehead, and ten days later the same sensation was felt

[1] Greeff, Bacteriological Investigations with Reference to the Origin of Sympathetic Ophthalmia, Archives of Ophthalmology, vol. xxii., 1893.

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in the eye itself. The bandage was removed from the right eye on the twentieth day. He could see nothing out of the left eye. At this time the right eye began to water very frequently, and soon became tired and painful and unfit for use. He noticed that when he held his injured eye shut he saw better with the other eye. Status: L. E., unirritable; corneal wound with incarceration of iris. Cataract. V = movements of hand at 1 metre. Projection uncertain, except outward. R. E., binocularly V = 6/9, and when left eye was closed, V = 6/6, and when the left eye was closed the field of vision in the right eye became wider. Schweigger has observed this condition several times, but there was always present some vision in the injured eye. Resection of the optic nerve was performed, and portions of the nerve were stained with Weigert's, Gram's, and Löffler's methods, but no organisms were discovered.

The road along which sympathetic irritation travels is a complex one. It is composed of the short ciliary nerves which come from the ciliary ganglion and of the nerves which enter into the composition of this ganglion, —namely, one from the nasal branch of the ophthalmic, another from the third nerve, and still another from the sympathetic. Among these the nerves which have most to do with the sympathetic irritation are the fibres derived from the fifth and the sympathetic. It is evident, then, that the irritation must pass along an eccentric course before it reaches the second eye.

TIME.

The time that elapses between the reception of the injury and the outbreak of the sympathetic affection in the other eye is an important factor in the history of sympathetic ophthalmia, and, while we may consider from three to eight weeks as the most likely limits within which the sympathetic inflammation makes its appearance, there have been not a few cases reported in which the interval was much shorter. Gunn[1] reports a case in which the sympathetic inflammation broke out in fourteen days. Iritis followed a cataract operation, and the pupil was completely closed with a thick exudate. Discission was performed eight weeks later, and fourteen days afterwards the other eye became inflamed. I do not think, however, that it would be illogical in this case to trace the sympathetic inflammation back to the first operation. It is more than probable that the first operation was the exciting cause.

Vignaux[2] reports a case in which the interval was ten days; but the reasons for this assumption are based entirely upon the history, and, as the patient was not seen till ten months after the injury, I am inclined to agree with Schirmer in regarding the interval of time as uncertain.

Mooren[3] reports four cases; in two of them sympathetic ophthalmia

[1] Gunn, On Sympathetic Inflammation of the Eyeball, Royal London Oph. Hosp. Rep., xi., 1886.

[2] Vignaux, De l'Ophthalmie sympathique et spécialement de son traitement par l'énucléation, Paris, 1877.

[3] Mooren, Ophthalmiatrische Beobachtungen, Berlin, 1867.

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appeared on the fourth day, in the other two on the sixth day. The first two certainly lacked important features peculiar to sympathetic ophthalmia, while in the last two cases both the supposed exciting eyes had passed through an attack of irido-chorioiditis spontaneous in character: so there is no good reason for not believing that the inflammation which affected the second eye in both cases was a spontaneous one.

O. Becker[1] reports a case in which sympathetic neuro-retinitis appeared ten days after the injury of the first eye, which latter had been destroyed by panophthalmitis. The case, however, is open to objection, as the patient was suffering with tetanus, and doubtless general infection was present.

Barrett[2] reports what seems to be a genuine case, in which sympathetic ophthalmia developed on the fourteenth day and the eye was destroyed.

Regarding time as an important feature in the history of the disease, the diagnosis will be freer from doubt if we consider three weeks as the earliest date for the outbreak of the sympathetic disturbance.

There is nothing but uncertainty in fixing the latest point of time for the outbreak of sympathetic ophthalmia. As a rule, the injured eye is completely blind before the outbreak of the sympathetic disturbance, though this is not necessary, for cases have been frequently reported in which the injured eye retained a certain amount of vision throughout, and at the end was the better eye of the two. I should say that the usual limit has been passed when the sympathetic inflammation fails to make its appearance before the end of the fourth month after the injury. The shorter the interval the more doubt surrounds the diagnosis, and the longer the interval the more uncertain the diagnosis becomes.

Lee[3] reports a case in which a man had lost his eye in early youth. The eye had atrophied and had given no trouble for forty-seven years, but for the past six months he had been troubled with lacrymation in it. In the second eye there was slight pericorneal injection, the pupil reacting badly to atropine. There was also slight clouding on the posterior surface of the lens. These symptoms disappeared on the enucleation of the primarily affected eye.

Knapp[4] reports a case which occurred forty-five years after the injury, and Chisolm[5] mentions one in which the sympathetic disturbance manifested itself thirty-five years after the injury. In Alt's[6] table twenty-two and three-quarters per cent. occurred between one year and ten years, twelve per cent. between ten years and twenty years, and thirteen and one-third per cent. between twenty-three and sixty years. I may say, however, that

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[1] O. Becker, Ueber die Entstehung der sympathischen Ophthalmie, Archiv f. Psychiatrie und Nervenkrankheiten, 1882, S. 250.

[2] Barrett, Australian Medical Journal, 1891, vol. xiii. p. 341.

[3] Lee, British Medical Journal, 1885, vol. ii. p. 397.

[4] Knapp, Archives of Ophthalmology, vol. i.

[5] Chisolm, New York Medical Journal, vol. xxi. p. 193.

[6] Alt, Archives of Ophthalmology and Otology, 1876.

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I do not think that an atrophic eyeball which is absolutely free from irritation, inflammation, and pain can give rise to an inflammation in the other eye, and that so long as the atrophic eye remains insensitive to moderate pressure the danger of sympathetic ophthalmia amounts to nothing. These cases may be regarded as atypical forms of the disease, and while there is not the same doubt about the diagnosis here as in those cases in which the sound eye takes on sympathetic inflammation in the first or second week, still unusual conditions must be present to cause such irregularity, conditions which I think ought to separate this class of cases from those in which the outbreak of the sympathetic affection occurs within the limit just mentioned.

PATHOGENESIS.

The pathogenesis of sympathetic ophthalmia has been made the subject of considerable experimental work, which, on the one hand, has had the effect of stimulating interest and investigation in this quarter, and, on the other, while resulting in little that was positive, has led to the clearing away of much that was obscure and false.

Mackenzie[1] may be regarded as the pioneer among those who have sought to clear up the pathogenesis of the disease. He suggests three possible explanations in this connection:

"First, it is not improbable that the blood-vessels on the side of the injured eye, being in a state of congestion which attends inflammation, communicate to those of the opposite side with which they have connections within the cranium a disposition to the same state in which they themselves are.

"Second, the ciliary nerves also of the injured eye may be the means of conveying to the third and fifth nerves an irritation which may be reflected from the brain to the same nerves on the opposite side.

"I think, however, that the chief medium through which sympathetic ophthalmitis is excited is the union of the optic nerves. It is extremely probable that the retina of the injured eye is in a state of inflammation which is propagated along the corresponding optic nerve to the chiasm, and there the irritation which gives rise to inflammation is reflected to the retina of the opposite eye along its optic nerve."

This theory found general acceptance among ophthalmologists till the appearance of Heinrich Müller's[2] investigations some years later. Müller endeavored to find the channel of communication in the ciliary nerves. He draws his conclusions from the anatomical examination of three eyes which had been enucleated through fear of sympathetic disease in the fellow-eyes. Müller concedes the possibility of inflammatory transmission along the optic nerve, though the latter, he says, all the way from the retina up into the trunk, is in a condition of atrophy, so that it is not capable of

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[1] W. Mackenzie, loc. cit.

[2] H. Müller, Anatomische Beiträge zur Ophthalmologie, Archiv f. Ophth., 1858, 1, iv.

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conducting an irritation, or, in fact, any other process. Cutting through the optic nerve, then, will not lessen the chances of sympathetic trouble. The ciliary nerves, on the other hand, do not easily atrophy. The majority of eye-diseases attack the anterior part of the eye, and in consequence the ciliary nerves, from their location, would be more exposed to irritation. And when the inflammation of the second eye makes its appearance under the garb of irido-chorioiditis, as it frequently does, it is far more logical for us to assume that the inflammation was brought about through the ciliary nerves rather than through the optic nerves. It is not improbable, he says, that the ciliary nerves exercise some direct influence upon the nutrition of the retina and optic nerve. Every cyclitis, whether of spontaneous or of traumatic origin, whether it made its appearance in the beginning as cyclitis or developed into the latter, always keeps up a more or less persistent irritation of the ciliary nerves. It makes no difference whether the phenomena of irritation are due to a genuine cyclitis or to any influence which interferes with the action of the ciliary body, such as stretching or tearing, calcareous products in the ciliary region, partial detachment of the ciliary body; under all circumstances the sympathetic disturbance which results rests upon the same principle, irritation of the ciliary nerves, together with an influence which affects nutrition, secretion, and accommodation.

Müller's observations had the effect of diverting the current of opinion from Mackenzie's theory, and the belief became wide-spread that the ciliary nerves alone were concerned in the transmission of the disease from the primarily affected eye to the second eye, and thus was started the ciliary-nerve theory. Müller found an ardent supporter in von Graefe, and, I might say, in the majority of ophthalmologists up to within twenty years ago.

The fact (if it be a fact) that changes in the structure of the ciliary nerves are not often seen is, as I have said elsewhere, a point in favor of the ciliary-nerve theory, though many of the opponents of this theory think differently. They regard the lack of pathological changes in the ciliary nerves as proof positive of their non-participation in the inflammation, having the idea that the inflammation travels along the ciliary nerves from one eye to the other, producing structural changes throughout its course. This, however, is very improbable. The only hypothesis upon which the ciliary-nerve theory can stand is the assumption that the inflammation in the injured eye calls forth a functional irritation of the sensitive ciliary nerve-fibres, and that this influence finds its way by reflex action to the fellow-eye. Pathological changes in the ciliary nerves would interfere with the propagation of such an irritation or influence; their soundness, then, speaks for and not against the theory. It should always be remembered that the ciliary nerves of one eye transmit influences or conditions to the fellow-eye through reflex action, and not, as is possible in the case of the optic nerves, through direct contiguity.

Since Müller's day observations in this direction have multiplied, and

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structural lesions in the ciliary nerves have been observed by Kraus,[1] Uhthoff,[2] Schmidt-Rimpler,[3] and others. Berger[4] examined fourteen eyes which had been enucleated on account of sympathetic ophthalmia, and in nine cases he observed inflammatory changes in the ciliary nerves within the supra-chorioidal space: these changes can go backward by way of the sclera as well as forward, for he found an enormous accumulation of round cells all around the blood-vessels, which accompany the ciliary nerves even up into the cornea. Berger[4] mentions Poncet, Ayres, Alt, Bolling, and others as having met with similar changes in the ciliary nerves. So far as my own experience goes, I have not noticed the absence of inflammatory changes about the ciliary nerves. These eyes have, as a general thing, undergone violent inflammation in every part, and there is absolutely no reason why the ciliary nerves should escape.

Mooren and Rumpf[5] published the following experiments, which they regarded as evidence in favor of the ciliary-nerve theory. They drew out a portion of the iris in a rabbit and dropped upon it a few drops of essence of mustard. Violent injection of this eye was seen, associated with, at first, anæmia in the second eye, followed by injection. If they treated the iris repeatedly with the mustard essence, the hyperæmia lasted an hour, and finally, on the following day, hyperæmia of the iris and clouding of the aqueous humor were noted. If they substituted ether, anæmia of the first eye and hyperæmia of the second eye were observed. There is, however, as Deutschmann says, too much confusion of hyperæmia with inflammation to allow us to look upon these experiments as especially convincing from any point of view. In other words, there is scarcely any pathological distinction between hyperæmia and inflammation, a difference of condition upon which pathologists of the present day insist.

The subject of sympathetic ophthalmia was ably discussed by Mauthner,[6] but from the unsettled tone of the work the Vienna professor leaves such of his readers as are devoid of a better insight into its history with as vague an idea as possible of the pathogenesis of sympathetic ophthalmia. In speaking of the possibility of transmission through the blood-vessels, Mauthner thinks that the inflammatory process, starting in the chorioid of one eye, might be carried over through the chorioidal vessels to the ophthalmic artery, from this point to the internal carotid, and thence to the circle of Willis. The inflammation then passes along the anterior arch of this circle into the opposite ophthalmic artery, and so into the chorioidal region of the second eye. He seems to think, however, that this is the

[1] Krause, Archiv für Augenheilkunde, 1882, x. 629.

[2] Uhthoff, Beiträge zur pathologischen Anatomie des Auges. Seltener Befund an zwei Ciliarnerven, Archiv für Ophthalmologie, xxix., 1882.

[3] Schmidt-Rimpler, Beitrag zur Entstehung der sympathischen Ophthalmie und Discussion, Heidelberger Congress, 1871.

[4] Berger, Beiträge zur Anatomie des Auges, Wiesbaden, 1887.

[5] Mooren und Rumpf, Centralblatt für die medicinischen Wissensch., No. 19, 1880.

[6] Mauthner, loc. cit.

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least tenable of the theories, and consequently lays upon it very little stress. In discussing the optic- and ciliary-nerve theories his arguments are characterized rather more by diplomacy than by a pronounced individual opinion, for he commits himself to neither of the two. "We have, on the whole," he says, "no right to ask whether the sympathetic affection is transmitted along the optic nerves or along the ciliary nerves; nor can we ask whether the transmission takes place along one path more frequently than along the other, for the transmission may be effected in both ways."

Not long after, the old Mackenzie theory was revived by Horner and Knies,[1] and, while convincing some, it had the healthy effect of stimulating inquiries in this, probably the darkest, field in eye pathology. The revival of this theory was the result of an observation by Knies[1] in the case of a girl nineteen years old affected with iritis serosa. In the left eye there were deposits about the size of pins' heads on the membrane of Descemet. Nothing of the fundus could be seen with the ophthalmoscope. She counted fingers at a distance of six feet. In the right eye there were slight deposits in the lower half of the cornea about Descemet's membrane. The corpus vitreum was clear. The papilla was markedly red and somewhat foggy. There were no synechiæ on either side. The patient was seized with violent bronchitis, and died soon after from gangrene of the lungs. From sections made the following was noted. The entire iris was infiltrated with round cells, as were also the ciliary body and the chorioid as far as the optic nerve. The latter showed neuritis and cell-infiltration, which continued into the orbit and as far as the chiasm. At this point the pial sheath of the nerve was markedly infiltrated. This condition was present in both eyes, and in consequence the idea suggested itself that between the two eyes, affected alike, a channel of communication through the medium of the optic nerves might exist, particularly as sympathetic inflammation usually makes its appearance under the garb of iritis serosa and premonitory papillitis.

Knies concluded that the inflammation had travelled from the originally injured eye up its optic nerve to the chiasm, across the latter to the other optic nerve, and from there down to the fellow-eye. Horner and Knies thought that they had found still further foundation for their theory when, after injecting a colored fluid into the subarachnoid space of one nerve, the fluid was found to have forced its way through the chiasm into the other nerve-sheath. "The pathologico-anatomical fact and the experiments will at least justify us," says Horner, "in explaining the transmission of the sympathetic process as having occurred through the lymph-spaces, and we ought now to abandon the uncertain field of reflexes, vaso-motor disturbances, and neuropathic inflammations."

I cannot agree with Knies in thinking that sympathetic ophthalmia

[1] Horner und Knies, Correspondenz-Blatt für Schweizer Aerzte, ix. Jahrg., 1879, No. 21; Ueber sympathische Augenentzündung, Festschrift für Prof. Horner, Wiesbaden, 1881.

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most frequently makes its appearance under the garb of serous iritis; and, while the case reported is a valuable one, he is not justified in concluding that the iritis passed from one eye to the other by way of the optic nerves, simply because the pial sheath of the optic nerve on both sides was infiltrated with cells. There was here a clear history of general infection; and, furthermore, serous iritis is not an uncommon disease, and in most cases it is dependent upon constitutional infection of some kind or other, under which circumstances, no doubt, the optic nerve on both sides would show evidences of inflammation as well as both irides. There is not a particle of reason here for not believing that the inflammation first attacked the iris in both eyes and involved the optic nerve secondarily.

In 1881 the subject was given still further impetus by the researches of Snellen,[1] Berlin,[2] and Leber.[3] These three advanced the opinion that the inflammation was of parasitic origin, and hence that the disease in the second eye must rest upon an infectious basis. They all agreed as to the nature of the ophthalmia, though they did not entertain the same opinion as regards the mode of transmission.

Snellen held that it was a specific inflammation, metastatic in nature, where the organisms were peculiarly adapted to the chorioidal tissue and were transmitted through the lymph-spaces of the optic nerve; and he went on to say, "if this theory of infection is the true one, the only path for the transmission of the organisms is the optic nerve. The ciliary-nerve theory is devoid of anything like convincing proof."

Berlin contended that a portion of the inflammatory products of the first diseased eye was taken up into the general circulation. These products can remain anywhere stationary in the organism without further development, simply because they do not find the conditions suitable for their nutrition. If, however, they get into the capillary region of the uveal tract of the other eye, they there find circumstances analogous to their mother soil, and they develop and give rise to inflammation.

Berlin's hypothesis, that the inflammation is a specific metastatic one and transmitted through the blood-vessels, opens up the possibility of inflammatory appearances in other parts of the body,—a condition, however, which is never present. It is the other eye alone which becomes involved. Such an hypothesis, then, is tenable only if we suppose that the nutrition and circulation in the eye differ from those of the rest of the body, that the eye possesses unique conditions for the growth of lower organisms, and that these organisms, in consequence, can attain their growth in the second eye, and nowhere else in the body. We have no right to assume this peculiarity for the circulation in the eye in contradistinction to that of the rest

[1] Snellen, Discussion on Sympathetic Ophthalmia, Trans. Internat. Med. Cong., London, 1881.

[2] Berlin, Volkmann's Sammlung Klinischer Vorträge, in Verbindung mit deutschen Klinikern, 186.

[3] Leber, Archiv für Ophthalmologie, xxvii. 1.

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of the body. The mere fact that the other eye alone is attacked justifies us in concluding that the cause must be a local one. By far the greater number of sympathetic eye-troubles are caused by the entrance of foreign bodies. Clinical experience shows us every day that wounds resulting from infectious foreign bodies cause the most violent inflammations, and that, on the contrary, wounds that are aseptic result in comparatively little disturbance. It may be well here to refer to some experiments [1] of my own, reported a few years ago, which have a bearing upon this aspect of the subject. After sterilizing a cataract-knife, I made an incision at the upper border of the cornea of a rabbit about half an inch below its junction with the sclera. I passed the knife into the iris and cut upward entirely through the corneo-scleral junction as far as the ciliary region, and in several instances into the latter. The cornea healed up promptly, as did also the iris, the healing process in the latter being exactly similar to that after a successfully performed iridectomy. The media remained clear throughout, and not a symptom of cyclitis followed the operation. Naturally, for thirty-six hours after the experiment there were some slight photophobia and lacrymation; but these soon passed away.

Leber has shown conclusively that aseptic foreign bodies may remain in the eye indefinitely without giving rise to inflammatory symptoms. Knapp [2] has performed a series of experiments which have similar weight in this connection. Leber [3] expresses himself unreservedly in favor of the infectious origin of sympathetic ophthalmia, and thinks that the optic nerve is the channel of communication, and hence that the disease spreads through continuity and contiguity of tissue. In typical cases of sympathetic irido-chorioiditis we have a condition of septic infection coming from without, so that the disease to a certain extent resembles the erysipelatous process.

So far as I can learn, Maats,[4] under Donders's direction, first made experiments with the idea of producing sympathetic ophthalmia in animals. In several rabbits there was produced a penetrating wound of the ciliary region, in others a foreign body was introduced into the eye and allowed to remain there, and in a third series a thread was drawn through the eye. Violent reaction followed in every case, showing itself generally in cyclitis. Recovery occurred in some of the animals, exudation into the vitreous body and lens and clouding were noticed in others, and in two cases there was phthisis bulbi. Maats experimented for ten weeks without obtaining anything but negative results. The eye which was not experimented on remained perfectly healthy, and the ophthalmoscope failed to reveal the slightest indication of sympathy. The same experiments were afterwards repeated by Snellen and Rosow, but without any positive results.

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[1] Randolph, A Contribution to the Pathogenesis of Sympathetic Ophthalmia, Archives of Ophthalmology, vol. xvii., No. 1.

[2] Knapp, Archives of Ophthalmology, vol. xv.

[3] Leber, Archiv für Ophthalmologie, xxvii. 1.

[4] Maats, quoted by Mooren, Ueber sympathische Gesichtsstörungen, Berlin, 1869.

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Of all the experimental work on this subject, that of Professor R. Deutschmann [1] has probably attracted the most wide-spread attention, and his results have been regarded by many ophthalmologists as settling the question beyond a doubt. The earlier experiments of Deutschmann were made with the spores of the Aspergillus fumigatus, and consisted in making repeated injections of a suspension of these spores into the corpus vitreum of a rabbit's eye. In this manner he produced sympathetic ophthalmia in the other eye, the inflammation showing itself not only in a neuritis optica, but also in an inflammation of the chorioid extending up into the ciliary body. Under the supposition, however, that the inflammation propagated in this way was the effect of chemical irritation (inasmuch as the spores of the aspergillus did not spread and no other organisms could be found near by), he injected croton oil into the eye of the rabbit, with a similar result. In all the successful experiments the pathologico-anatomical examination showed purulent infiltration of the infected eye, intense papillitis, and infiltration of the optic nerve and its sheaths; this infiltration passed up the optic nerve to the chiasm, spreading in a more or less moderate degree over the pia mater of the base, and then passed down into the second optic nerve, increasing in intensity as it neared the second eye. As a general thing he found the orbital tissue intact, except where he unavoidably infected the subconjunctival tissue at the inoculation. From these results he concluded that from an anatomical point of view the optic nerve and its sheaths in the case of a rabbit resemble those of a man, and in this respect behave alike when inflamed, certainly when the agents producing the inflammation are of a chemical nature.

Particles of copper and iron, even when aseptic, if introduced into the eyeball, can bring on inflammation by simply lying in contact with the delicate tissue, but such an inflammation generally remains circumscribed, and if it shows any tendency to spread it indicates that pathogenic bacteria passed in with the particle of metal. Inasmuch as foreign bodies which enter the eye in the case of man are infected, and as a chemical irritation is rarely, if ever, concerned in the production of sympathetic ophthalmia in human beings, he resorted to the pus organism for the inoculating material. He chose this organism because he thought from its wide-spread existence it would be more likely than other organisms to be concerned in the production of sympathetic ophthalmia. The staphylococcus pyogenes aureus and albus of Rosenbach were the organisms used. He injected a dilute suspension of these organisms into the corpus vitreum of the rabbit's eye, and produced generally chronic irido-chorioiditis with purulent infiltration of the corpus vitreum. He injected from one to two drops of the suspension. The infected eye usually underwent phthisis bulbi. Sometimes rupture of the eyeball occurred, though this termination he tried to avoid. Twelve hours after the injection of the micrococci, and

[1] Deutschmann, Archiv für Ophthalmologie, xxviii., xxix., xxx.

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sometimes earlier, if the media were moderately clear, Deutschmann observed a marked swelling of the optic papilla. In all these cases where such a condition was seen the microscopic examination would reveal abundant infiltration of the papilla with pus-cells, and this condition was also seen in the adjacent chorioid and vitreous body. When the experiment was successful, inflammatory changes would be discovered with the ophthalmoscope in the optic nerve of the second eye, and generally at a period ranging from five or six days to two or three weeks. The papilla of the sympathizing eye would be very red and prominent, and the blood-vessels dilated and tortuous. The edge of the papilla was swollen, and the substance of the papilla cloudy, so that the vessels running over it would appear veiled. The adjacent retina was cloudy, and the iris rigid. The pupil was narrow and irregular, but there were no well-marked posterior synechiæ. The animals died of general infection before the outbreak of a typical cyclitis. There was no meningitis present. He thought the premature death of the animal was the reason why sympathetic disease failed to appear in the second eye; in other words, the organisms did not have time to reach the second eye. If, then, he could shorten the path which the organisms took in their journey to the anterior portion of the uveal tract of the second eye, he would be able to produce an iritis before the death of the animal, and would have proved that had the animal lived long enough a sympathetic iritis would have developed itself.

For this purpose he made the following experiment. After a tenotomy of the superior rectus muscle, he passed in a delicate blunt instrument and pulled forward the optic nerve, which he severed as close as possible to the optic foramen. Into the cut end he injected a very small quantity of a suspension of the staphylococcus aureus, tied up the cut end to prevent the escape of the fluid into the orbit, and then replaced the parts. On the third day he established the existence of iritis with hypopyon, and the microscopic examination showed purulent infiltration of the iris and ciliary region. This proved that the micro-organisms, when they have time to spread in the sheaths of the optic nerve, pass down into the eyeball, causing inflammation all along their course and involving the entire uveal tract of the second eye.

There seem to be two routes which the organisms take on leaving the optic-nerve end of the second eye: they may pass out from the papilla through the chorioid into the iris; and, when the chorioid is not so much affected, they pass rapidly forward through the vitreous body to the zonula, and thence to the posterior surface of the iris and ciliary body. Deutschmann found the orbit in these experiments absolutely intact. The microscopic examination showed inflammatory changes not only in the infected eye, but also in the optic nerve of this eye, and these changes were found throughout the nerve up to the chiasm, and in the chiasm and down the other optic nerve to the fellow-eye, whence he concluded that the optic nerve and its sheaths represent the road over which sympathetic ophthalmia travels.

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The organisms, Deutschmann says, work their way forward against the lymph-stream that flows from the brain down through the sheaths of the optic nerve, and they make progress by reason of a certain impetus which comes from their own growth, as well as from their power of spontaneous movement. In this manner they reach the base of the brain, where they are swept down by the lymph-stream into the sheaths of the opposite optic nerve and thus reach the second eye: this movement on the part of the lymph-stream, he thinks, explains why the organisms do not spread themselves over the base of the brain and produce meningitis. He found no changes of any significance in the ciliary nerves, and does not think these nerves are concerned in the production of the disease.

In 1884 Alt[1] contributed the following account of some experiments made on animals. After drawing a thread saturated in croton oil through the optic nerve of a rabbit's eye, Alt noticed on the fourth or fifth day a neuro-retinitis in the other eye. This neuro-retinitis disappeared after a week. When he injected a putrid solution into the eye of a rabbit, panophthalmitis followed, and at the same time no sympathetic process was noted in the other eye. He then injected a few drops of an infusion of Abrus precatorius into the eyeball. The animals died generally in three or four days. One of the animals showed on the third day a well-marked neuritis, which disappeared in a few days. On injecting some of this infusion into the left eye of a rabbit, panophthalmitis resulted, and on the third day episcleral injection in the fellow-eye was observed. The iris became hyperæmic and swollen, the pupil was narrow and contained exudates. Later a genuine plastic iritis followed. The pathologico-anatomical examination revealed croupous exudation in the coats of the infected eye, together with infiltration of the papilla. He found the orbital tissue which surrounded the eyeball and the optic nerve also infiltrated. In the second eye Alt found a croupous exudation, neuritis, chorioiditis, and small-cell infiltration of the sheaths of the optic nerve. The ciliary nerves were sound. Alt concludes that in this latter case the inflammation in the second eye passed over directly from the infected eye, and that the track of this inflammation was along the optic nerve and its sheaths.

Two years later Gifford[2] attempted to confirm Deutschmann's work by a similar series of experiments. He performed seventeen experiments with the pus organism, and in not one was he able to produce a sympathetic inflammation in the other eye. In the infected eye violent inflammation invariably followed the injection, and the eyeball eventually underwent phthisis. In three cases panophthalmitis and rupture of the globe happened. The microscope usually revealed a purulent exudate filling up the vitreous body, small-cell infiltration of the retina and chorioid, and the physiological excavation filled with an exudate. He found the

[1] Alt, American Journal of Ophthalmology, No. 4, 1884.
[2] Gifford, Archives of Ophthalmology, vol. xv., 1886.

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lymph-spaces of the central canal of the optic nerve and also the lymph-spaces around the smaller vessels stopped up with leucocytes for a short distance beyond the eyeball, and next to the eyeball inflammation of the pial and dural sheaths was seen. The orbital tissue near the eye was markedly infiltrated. In a few cases there was a slight inflammatory exudate in the intervaginal space near the eyeball. The second eye remained normal in every case, though mention is made of a very slight hyperæmia of the papilla occurring in two cases and disappearing completely in a few days.

Gifford found organisms in the vitreous body of the first eye, located generally just in front of the papilla and behind the zonula, and he was never able to find them beyond the bottom of the physiological excavation, nor to demonstrate any round-cell infiltration of the central canal, the sheaths of the optic nerve, or the orbital tissue. Failing, then, to confirm Deutschmann's experiments with the pus organism, Gifford instituted a new series of experiments, using the anthrax bacillus. Out of twenty-five experiments he succeeded in obtaining a positive result in three cases, and in these three cases he found the bacilli generally in the perichorioidal space of the second eye.

A drop of water containing a particle from the surface of an old anthrax culture was injected into the vitreous body near the papilla, above and a little to the outer side. The next day the optic-nerve excavation was filled with an exudation which stretched out in the direction of the scleral wound. The left eye remained normal. The exudation continued to increase in the inoculated eye till the third day, when it appeared to diminish. The other eye remained normal, and the rabbit showed no general symptoms till nearly three weeks had elapsed, when it died of general infection. The microscopic examination showed bacilli sparingly present in all the blood-vessels. In the inoculated eye the bacilli in the vitreous body nearly all showed signs of disintegration, this condition manifesting itself by the difficulty in staining the bacilli. In the central optic canal the bacilli stained irregularly; in the smaller vessels, however, they were very numerous and were well stained. The bacilli extended along the vessels to the posterior part of the orbit, and these were the important points. The bacilli were also present in the cranial cavity on the surface of the pia covering the cranial portions of both optic nerves and the chiasm; also in the intervaginal space of both nerves, increasing in quantity towards the eyeballs to the point where the space begins to narrow, whence the line of bacilli tapered rapidly out and disappeared in the right eye, back of the perichorioidal space, and in the other eye the line of bacilli could be clearly traced for a short distance into this space. The inner layers of the dural sheath were densely infiltrated with bacilli, particularly near the globes. They were free on the surface of the pial and arachnoidal sheaths. In the subvaginal space there were more bacilli in the second eye than in the first, though the difference was not great.

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In the second experiment the papilla for the first three days was concealed by an exudation, just as in the first case. This condition persisted till the animal was killed. Microscopic examination showed bacilli and exudation in the vitreous body and along the central vessels, also in the supra-vaginal space posterior to the point where the central vessels leave the nerve. In the cranial cavity in front of the chiasm and between the optic nerves bacilli and exudation were to be seen. Between the sheaths and in the inner layers of the dural sheath the bacilli were present in larger quantities than in the first case, and there was about the same quantity in each. Along the central vein the bacilli were lying in such masses that in making sections, Gifford says, they were displaced by the knife and appeared as though within the walls of the vessels; but, with this exception, he says, in no part of eye, nerve, orbit, or chiasm were fungi to be found in the vessels. This makes it probable, he thinks, that no general infection had taken place when the animal was killed.

The third experiment resembled in its general features the first two, though the animal died of meningitis. No abnormal condition of the fellow-eye was ever observed with the ophthalmoscope, and microscopically the papillæ were normal. Gifford concludes from his experiments that micro-organisms can in rabbits be carried by the lymph-stream from the vitreous body of one eye to the space around the chorioid in the other, and that the most direct and open path from one eye to the other, and that taken by the anthrax bacillus, does not lie in the substance of the nerves themselves, nor in their sheaths, but, leaving the first optic nerve with the vessels, passes through the orbit into the cranial cavity, and thence *via* the subvaginal to the supra-chorioidal space of the second eye. Gifford, on the whole, seems to incline to the belief that the disease is of infectious origin, though he confesses that his experiments do not in any way strengthen Deutschmann's position.

Mazza[1] experimented also upon rabbits and guinea-pigs, but reports only failures from Deutschmann's point of view. Nor did this observer notice any change in the second eye with the ophthalmoscope. In those animals that had died of meningitis, cocci were to be found in the optic nerve and its sheaths.

About this time I[2] commenced a series of experiments in the Pathological Laboratory of the Johns Hopkins University, on rabbits, for the purpose of controlling the results obtained by Deutschmann. My experiments consisted in injecting a suspension of the staphylococcus aureus into the vitreous body. The instruments employed were always thoroughly sterilized previous to the operation. About two minims of the inoculating fluid were injected. After fixing the eye with forceps, I pulled the eye forward and introduced a hypodermic needle into the vitreous body at a point

[1] Mazza, Ueber experimentelle sympathische Ophthalmie, VII. Internationaler Ophthalmologen-Congress zu Heidelberg, 1888.

[2] Randolph, loc. cit.

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half an inch posterior to the ciliary region. The organisms were obtained from a furuncle, and were identical with the staphylococcus aureus of Rosenbach. I may be permitted to quote here the histories of two of the cases that were mentioned in the report of my experiments.[1]

CASE I.—Inoculated a small black dog in the right eye, injecting two minims of the suspension in sterilized water. Slight clouding of the vitreous body was observed in a few hours. The next morning the media were so clouded that it was impossible to get a view of the fundus. Irido-cyclitis ensued, followed by panophthalmitis and rupture of the eyeball, the latter occurring on the third day after inoculation. Healing of the inoculated eye was prompt. I examined the fellow-eye with the ophthalmoscope every day, but never detected anything abnormal. The animal was killed on the twenty-fourth day, and the eyes were carefully dissected out with their optic nerves and the chiasm. Phthisis bulbi was the condition of the inoculated eye, but no atrophy of the nerve was present; indeed, there was no microscopic difference in the appearance of the two nerves. Microscopic examination showed the inoculated eye densely infiltrated. Everywhere throughout the uveal tract, retina, and vitreous body lymph-cells were abundant. The papilla was very much swollen and was densely infiltrated with round cells. This cell-infiltration extended on up the nerve, involving not only the trunk of the nerve itself, but also the sheaths and the intervaginal space. This infiltration decreased somewhat in intensity on nearing the chiasm, and at this point it was hardly possible to say with certainty whether the physiological number of nuclei had been overstepped. There was certainly nothing that would be regarded as pathological in that part of the chiasm which is continuous with the nerve of the healthy eye. Examination of the second eye and its nerve revealed a perfectly normal condition of the parts. There was no cell-infiltration of the orbital tissue adjacent to the inoculated eye, nor of any portion of the brain touching upon the optic nerves and commissure. The most careful examinations failed to show the presence of organisms.

CASE II.—Inoculated a brown puppy, five months old, in the right eye; irido-cyclitis, followed by rapid phthisis bulbi. The eye did not rupture. Twenty-six days after the inoculation I observed in the left eye a clouding of the cornea. On a close examination by oblique illumination I became convinced that the clouding was not superficial, but, on the contrary, deep-seated; in other words, that there existed an interstitial keratitis. The surface of the eyeball was entirely free from wound or abrasion; nor, indeed, was there any external cause visible. A cause from without would probably have given rise to a superficial inflammation, but here the superior layers of the cornea were intact. Extrinsic causes do not give rise to inflammations of the cornea limited to the parenchyma. The cause here was plainly intrinsic, and I naturally supposed that I had before

[1] Archives of Ophthalmology, vol. xvii. pp. 188–213.

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me a typical case of sympathetic ophthalmia, in which the organisms, after traversing the uveal tract, had passed over from the iris to the membrane of Descemet and there set up inflammation. In two or three spots on this membrane there were small white deposits. If, however, the organisms had taken this track, there must be present also an iritis and a chorioiditis. Owing to the corneal clouding, it was impossible to ascertain the condition of the fundus; but the iris was in every respect normal, acting promptly. The dog was kept under observation for forty-eight hours, and, no new symptoms having developed, was then killed. The microscope showed throughout the inoculated eye dense round-cell infiltration, and neuritis optica extending about half an inch from the papilla, but from this point up the optic nerve to the chiasm and down the other optic nerve no pathological change was to be seen. As regards the second eye, and particularly the chorioid, ciliary body, and iris, I found not the faintest trace of an inflammatory process other than the keratitis, and I should say that, with the exception of the cornea, the eye was perfectly normal. The keratitis, then, was a spontaneous process, entirely distinct in its origin from the causes which resulted in the destruction of the other eye. A close examination for micro-organisms failed to reveal the presence of the latter.

The records of these two cases are fairly typical of the general history of all fifteen of the experiments performed on dogs. The other thirteen cases differed in but few particulars from the above, certainly in nothing that could be regarded as throwing any additional light upon the subject. I selected dogs because I thought they would be less likely to succumb to a general infection than rabbits; and as regards this question of general infection, the whole history of the inflammation, as seen in my experiments, resembles more nearly what we see in man, for in only one instance did I observe anything like a constitutional effect, and that was in the last case reported. The trouble, in other words, remained local. The keratitis in this case was, in my opinion, a manifestation of constitutional disturbance brought about by the presence of micro-organisms in the blood.

My second series of experiments were made on rabbits, and numbered fifteen in all, and the results obtained differed in no essential points from those in the first series on dogs. I may add that all the cases were examined every day with the ophthalmoscope, but never was there anything abnormal observed about the second eye. The fundus remained unchanged, perfectly normal; not even did the blood-vessels take part by increase of size; in other words, there was no hyperæmia. I might mention, however, that in two cases in the first series there was noticed twenty-four hours after inoculation an enlarged condition of the retinal vessels, associated with a general redness of the papilla. This disappeared at the end of three or four days. It certainly could not have been due to the presence of organisms, for there was not the faintest trace of an inflammatory process in the fundus, and the corpus vitreum was always perfectly clear. It was, I think, a reflex engorgement of the vessels, due to the violent inflammation going

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on in the other eye, and there is no reason to believe that it could ever have developed into a condition similar to that of the first eye. Every one of the eyes in both series suffered with irido-cyclitis in its most typical form.

Three years later Limbourg and Levy,[1] of Strassburg, published the results of an exhaustive line of experiments. These observers inoculated twenty-five rabbits and seventeen guinea-pigs after the method of Deutschmann. The changes in the inoculated eye resembled precisely those observed in my own experiments. "As regards the second eye," to use their own words, "no changes were observed which could be looked upon as sympathetic, with the exception of a very doubtful hyperæmia of the fundus. The examination in every case for organisms was negative." Their work simply confirms my own.

As I have elsewhere remarked, it is surprising that the authors, after finishing such a valuable work, did not feel themselves justified in drawing any further conclusion than this: "It would seem, then, that the experimental investigation of this question is very difficult."

To the experiments of these different observers I may add those of Schirmer,[2] Greeff,[3] and Ulrich,[4] all seeking one end, that of producing sympathetic ophthalmia in the lower animals by the same methods employed by Deutschmann, Gifford, Mazza, and myself.

Schirmer[2] inoculated twelve rabbits with staphylococci obtained from a furuncle. In every case the inoculation was followed by a more or less severe purulent uveitis which ultimately led to phthisis bulbi. Two of the animals died of a general infection. In five cases there was perforation of the eyeball. In the case of the five animals that suffered no perforation of the eyeball after the inoculation, the eyes were in the beginning examined with the ophthalmoscope every day and kept under observation for several months, and in not a single instance did the second eye show the slightest objective changes; consequently he did not think it necessary to make any microscopic examination. Schirmer made three other experiments with a streptococcus, but obtained a negative result in each case, though the eyes were examined systematically for some months.

Greeff[3] injected a putrid solution into the vitreous body of three rabbits, and panophthalmitis followed, with rupture of the eyeball. No changes were visible in the second eye. The inoculated eye was enucleated a few weeks later, and, with a small portion of the nerve, was examined for organisms, but with a negative result. The aspergillus fumigatus was injected into the vitreous body of four rabbits. In two cases he observed a slight haziness of the fundus with tortuosity of the vessels and redness of the papilla, but these appearances were gone in two or three days. These

[1] Limbourg und Levy, Untersuchungen über sympathische Ophthalmie, Archiv für experimentelle Pathologie und Pharmakologie, xxviii., 1890.

[2] O. Schirmer, loc. cit.

[3] Greeff, loc. cit.

[4] Ulrich, Bericht der ophthalmolog. Gesellschaft, 1891.

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phenomena he agrees with me in attributing, not to the presence of organisms, but to a reflex overfilling of the vessels caused by the inflammation going on in the inoculated eye. He injected a suspension of the staphylococcus pyogenes aureus into the right eye of seven rabbits, and after a certain length of time an examination was made of the end of the optic nerve of the right eye and also of the left eye. The blood from several of the organs was also examined. One of the rabbits died of meningitis, two wasted away, and four lived. An examination in the first three cases revealed inflammatory changes in the second eye, and organisms were found in the end of the optic nerve and also in other organs of the body. Of the four rabbits that lived, one showed slight changes in the second eye, but no organisms were found in any of these cases. The fact could not be established that a direct migration of the organisms through the channel of the optic nerve and its sheaths had occurred. And, furthermore, organisms were found in the second eye in those cases only in which organisms were present in other organs of the body and thus had reached the second eye through the general circulation.

I may simply mention the fact that Ulrich's[1] experiments resulted in like manner; and, finally, reference should be made to the very recent work of Bach,[2] who after repeating the experiments of Deutschmann failed in any particular to confirm the results of that observer.

It is evident to every one that the solution through experiment of any question in pathology is valuable only when the various steps of the solution can be followed out by others. If, then, other observers repeat the original experiments, and with results similar to those of the author, the latter's conclusions have been verified and the question has been settled. An observer may hold the right view as to the pathogenesis of a disease, but if he attempts to prove that he is right by a series of experiments on animals there must be absolutely no inconsistencies between the steps of his work and his conclusions. In the first place, he must produce experimentally a disease which is identical with the disease in man. He must, in other words, find an animal in which he can produce the disease in question, and the disease should resemble in all its clinical features just what we see in man. It is needless for me to add that it is impossible to produce some diseases in some animals. There are some diseases which are common to both man and beast, but, as a rule, each kind of animal has its own peculiar class of diseases. So far as I can learn, there has never been observed a well-established sympathetic inflammation of *traumatic* origin in the eyes of any of the lower animals, and there seems to be no reason for believing that rabbits are different from other animals in this respect. And inasmuch as rabbits are so constantly and widely used for all sorts of experiments, they are necessarily under very close observation, so that incidental ocular

[1] Ulrich, loco citato.

[2] Ludwig Bach, Archiv für Ophthalmologie, Bd. xlii., 1, S. 241–276.

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affections would not be likely to escape the notice of a careful observer. I may say that, notwithstanding a connection of several years with a laboratory where there are hundreds of these animals used yearly, I have noticed only once an eye-affection in a rabbit, and that was a case of keratitis. Dogs, on the other hand, exhibit ocular symptoms very frequently in connection with all kinds of experiments. I once observed seven or eight cases of keratitis in dogs in whom portions of the thyreoid gland had been extirpated. Rabbits, then, seem to present peculiarities in their freedom from eye-diseases, certainly a noteworthy fact when we take into consideration man's susceptibility in this direction, or even that of the dog. For this reason I regard rabbits as unsuitable subjects for illustrating the eye-affections met with in man, and, as all the experiments have shown, peculiarly unfitted for the purpose of throwing any positive light upon the pathogenesis of sympathetic ophthalmia.

The great ease with which the fundus of the rabbit's eye can be studied in all its details, even by a novice in ophthalmoscopy, precludes the possibility of any mistake having been made by the various observers, who, with the exception of Deutschmann,[1] report absolutely negative ophthalmoscopic conditions in all their experiments. I would call attention to the difficulty of deciding in experiments of this character on rabbits, and even on dogs, whether a section of the nerve is normal or not, unless one has at hand a section from a corresponding point of the nerve which is known to be normal and with which the supposed pathological specimen can be compared. This is also true of the chiasm. In the first five experiments on dogs[2] I was at first convinced that I had a neuritis extending from the infected eye around to the fellow-eye by way of the chiasm. I submitted the section to skilled microscopists, and they agreed with me. The suggestion was made, however, that I kill a dog and study the normal condition of the part. I did this, and, to my surprise, found that the normal optic nerve of the dog was very rich in nuclei, which gave to the nerve the appearance of dense round-cell infiltration. It should be remembered that the chief distinguishing feature of a neuritis at this stage is the increase in the nuclear elements, and upon this we must mainly rely in making a diagnosis of neuritis. Changes in the nervous bundles themselves are not readily demonstrable features of a neuritis of three weeks' duration.

We must regard the number of nuclei, then, as in great measure deciding the existence of inflammation, and the fact that the physiological number of these nuclei, from their abundance, can easily deceive us and lead us to think that we are looking upon a pathological number,—this fact, as I have said before, necessitates comparing what is known to be a normal condition with the section under observation. In other words, longitudinal and transverse sections of the ball, optic nerve, and chiasm of

[1] Deutschmann, Archiv für Ophthalmologie, xxviii., xxix., xxx.
[2] Randolph, loc. cit.

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the eye to be examined should be compared with corresponding sections that are known to be normal. I have observed that normally the nuclei are more scattered and are much fewer in the neighborhood of the chiasm, while they are abundant at the intra-ocular end of the nerve, and are comparatively few in number in the chiasm. I regard these precautions as absolutely indispensable in the microscopic examinations of the parts just mentioned in experiments on dogs, and what I have said of the normal histology of the dog's optic nerve and chiasm is equally applicable to the rabbit. Unless these precautions be taken, then, a reasonable doubt must be cast upon the accuracy of a diagnosis of existing inflammation.

Again, in experiments of this kind too much stress must not be laid upon one result which accords with the theory of the experimenter. If among ten experiments only two give positive results, the chances are that the eight negative results represent the true state of the case, while the two positive ones may be accidents. The oft-repeated assertion, made by Deutschmann[1] and others, that one positive result proves more than many negative ones, is not correct. The fact that contaminations and mistakes may occur at every step in this kind of work justifies one in holding the very opposite of this view. One positive result in bacteriological work cannot prove a theory, and only when the positive results have been repeated over and over again, and that, too, by many observers, can we say that the theory has been proved.

In reading the account of Deutschmann's[2] experiments one cannot feel otherwise than astonished at the apparent simplicity of his work and at the perfect ease with which he solves the question. Nothing could be more reasonable than the infectious theory of sympathetic ophthalmia, if we base our conclusions on Deutschmann's experiments with rabbits. The frequency with which he obtained positive results is no less striking than the reasonableness of the theory, and it is this latter point, I think, which in great measure led the majority of ophthalmologists to set an exaggerated value on these experiments and to regard Deutschmann's conclusions as final. It is no wonder, then, that a work of such importance should be subjected on all sides to careful investigation, and, considering the number of positive results obtained by Deutschmann, it is natural that those who attempted to repeat the experiments of the latter should have anticipated little or no difficulty. In a more recent work of Deutschmann's[1] he states that out of thirty-five experiments he obtained sympathetic inflammation only twice, a fact which, for reasons that I have stated, leads me to consider these two positive results of very little value. His earlier experiments were attended with, comparatively speaking, uniform success. As I have stated in the foregoing pages, his experiments were repeated by numerous observers, and Parisotti[3] was the only one who ever observed anything like participation

[1] Deutschmann, Beiträge zur Augenheilkunde, Bd. i.
[2] Deutschmann, Archiv für Ophthalmologie, xxviii., xxix., xxx.
[3] Parisotti, X. Internationaler Medicinischer Congress, Bd. iv., 1890, S. 109.

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on the part of the second eye. Even in these positive results of Parisotti's there is every evidence that the rabbits were more or less under the influence of a general infection, and it is clear that this condition nullifies the special value of the eye-symptoms in the second eye, for here the inflammation is due to the presence of organisms throughout the entire circulation, and not to the fact that the organisms have selected the optic nerve as their path and by this way reached the other eye. As I have shown elsewhere, one can inoculate a rabbit at the root of the tail with the anthrax bacillus, and, if the animal remains alive long enough, these organisms will be found everywhere throughout both eyes, and if the vitreous body of one eye be inoculated with the anthrax bacillus, this organism can be detected in a few hours in the fellow-eye. Professor Meyer[1] was requested to look at what was thought to be a case of sympathetic ophthalmia which had been produced experimentally in a rabbit. He found an almost typical condition in both eyes, but on looking at the other rabbits in the same laboratory he observed exactly the same symptoms, although these latter had been used for an entirely different character of experiments, in which the inoculation had been made on parts of the body remote from the eye.

Gayet[2] reports a case of what he regards as sympathetic ophthalmia, which he produced in a rabbit by introducing into the anterior chamber a piece of tumor taken from the lacrymal sac. The inoculated eye became blind, and fourteen days later the other eye became inflamed and took on a violent keratitis. In my opinion this was another case of general infection which resembled one of my cases in a dog, and also similar to those that I have mentioned as occurring in dogs in whom portions of the thyreoid gland had been removed; and here I may add a very important point, and that is, that sympathetic ophthalmia does not take on the form of a simple keratitis. It may be well to add that Parisotti,[3] while he found organisms in the chiasm and in the optic nerve of the second eye, was never able to demonstrate their presence in the second eye itself.

When we take into consideration the results of the many who have attempted to produce the disease experimentally, we are forced to one conclusion, and that is, that the pathogenesis of sympathetic ophthalmia is not demonstrable through experiments on dogs and rabbits. It should be remembered that, with hardly an exception, those who entered upon the work of testing this question experimentally did so with preconceived notions as to the nature of sympathetic ophthalmia, notions which were more or less in accord with those of Deutschmann. There was then no disposition to break down or pick flaws in a work that seemed well-nigh faultless, but simply a desire to confirm and strengthen this work; so that it is all the more astonishing how completely Deutschmann's experi-

[1] Meyer, Bericht der ophthalmolog. Gesellschaft, 1891, p. 104.

[2] Gayet, Recherches anatomiques sur une ophtalmie sympathique expérimentale, Archives d'Ophtalmologie, t. x., 1890.

[3] Parisotti, loc. cit.

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ments have fallen short of verification, and I am inclined to the belief, in which others share, that he fell into serious errors of observation and interpretation. In addition to proving that rabbits and dogs do not have sympathetic ophthalmia, the results of the experiments of Deutschmann and of those who repeated them show conclusively that the pus organism plays no part in the production of the disease in these animals, and this is what we should be apt to expect if we consider the behavior of this organism when met with in the human eye. While panophthalmitis does not preclude the possibility of the occurrence of sympathetic ophthalmia, it is a well-known fact that sympathetic ophthalmia has been rarely known to follow a case of panophthalmitis, a disease in which the pus organism is present in large numbers. Both Leber and Deutschmann think that the reason the disease does not occur under these circumstances is that the fungi are destroyed by the suppurative process, and, in addition, the infectious contents of the eyeball are emptied when the latter bursts.

Gifford[1] thinks that this explanation is probably incorrect from observations made in cases of panophthalmitis in rabbits. In cases where the eyeball had ruptured, he made cover-glass preparations and agar cultures from the interior of the vitreous body, and succeeded in demonstrating the presence of organisms perfectly capable of developing. He found organisms equally active in eyes where the inflammation had passed its height, and where phthisis had set in without rupture. Gifford is of the opinion that the lymph-spaces are blocked up by the products of the inflammation, and that in this way, possibly, a mechanical obstacle is offered to the migration of the organisms. All three explanations are reasonable, and no doubt would explain all such cases were it not unlikely that the pus organism has anything to do with the production of sympathetic ophthalmia. I may mention in this connection that Schirmer,[2] Lawson,[3] and others report cases of the disease following panophthalmitis. There is, however, no doubt as to the rarity of such an occurrence. It seems fair, then, to conclude that the numerous experiments made on the lower animals with the pus organism only prove the immunity of the animals from sympathetic ophthalmia and the negative part played by the pus organism. In other words, these experiments have shed absolutely no positive light upon the pathogenesis of the disease.

As regards the discovery of organisms in enucleated eyes, especially in those cases in which sympathetic inflammation existed, there are several points to be considered.

1. The demonstration of organisms in eyes which have been the seat of traumatic inflammation is not a surprising thing, even though such eyes have given rise to sympathetic ophthalmia. Such a demonstration does not warrant the conclusion that these organisms have caused the sympathetic

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[1] Gifford, loc. cit.

[2] O. Schirmer, loc. cit.

[3] Lawson, Royal London Ophthalmic Hospital Reports, vol. vi.

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inflammation. Again, by far the greater number of investigators have failed to find bacteria, even in those eyes removed in cases of coexistent sympathetic ophthalmia, so that it is but just to conclude that the disease can occur without the presence of bacteria. This fact should lead us to set comparatively little value upon the discovery of organisms in eyes that have not given rise to sympathetic ophthalmia.

2. It would be well to call to mind the chain of proof formulated by Koch, that a specific micro-organism of an infectious disease must be present in all cases of the disease in such number and situation as to explain the phenomena of the disease, and must be capable, when inoculated in pure culture, of reproducing the disease.

The following case I reported in the *Archives of Ophthalmology* (vol. xxi., No. 3), and I quote it in full, as I look upon it as specially valuable from a bacteriological point of view. J. M., aged forty, farmer. While endeavoring to dig out a large stone deeply embedded in the earth, a piece of the pick broke off and flew into his right eye. This was on June 1, 1891. He continued with his work, though seeing very indistinctly with the wounded eye. He was not conscious of any pain at the time. He kept on regularly with his farming for three days longer, when pain and dimness of sight compelled him to rest. He came to the hospital on the fifth day. At this time he had only light-perception left in the wounded eye. The eye was exceedingly sensitive to the touch. He remained in the hospital three days, and left with directions what treatment to follow, and with the understanding that he should return in case the pain persisted. After being at home six weeks and suffering all the time, his good eye began to show signs of sympathy in photophobia, lacrymation, and dimness of vision, and according to his account he could not see a horse a hundred yards away. Before this he possessed unusually acute vision. His family physician treated him with blisters and cold applications to the eye, and his sight improved. August 13 he again came to the hospital, and the condition of his eyes was as follows. In the injured eye light-perception was gone; eyeball sensitive to the touch. At the point of the wound the sclera was injected, cornea clear, pupillary margin entirely bound down by adhesions to the anterior capsule. His good eye had a vision of 10/200. The anterior capsule showed abundant inflammatory deposits upon its surface, together with numerous evidences of old iritis all around the pupillary margin. Indeed, it was difficult to make out the fundus with any distinctness, on account of the general dirty appearance of the lens. He complained of great pain in the forehead and in the region behind the nose. He was persuaded to have the eye enucleated, and I prepared to make cultures from the vitreous body and anterior chamber. The eye was removed, and, after sterilizing with a hot knife a point at the corneo-scleral junction not far from the wound, I entered the anterior chamber with a spade knife that had been sterilized by passing it several times through an alcohol flame. I withdrew the knife and followed it with a platinum wire

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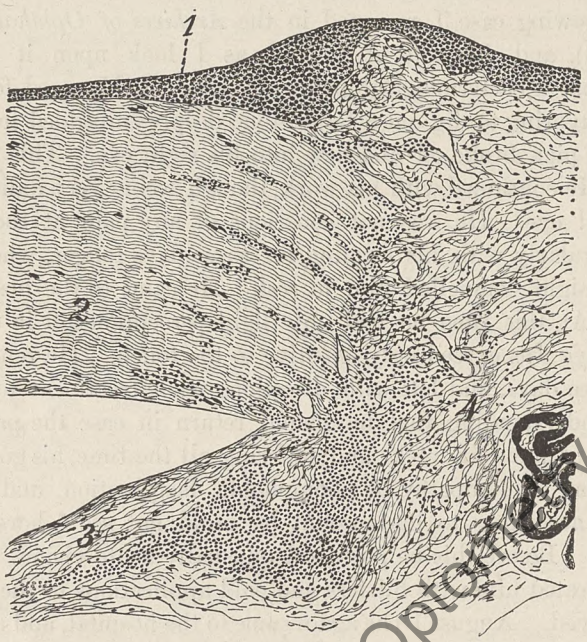
loop, which was stirred up in the anterior and posterior chambers, and also in the vitreous body, and smear cultures on agar were made, and also three Esmarch tubes. These tubes were subjected to the proper temperature in an oven for as long as ten days, and not a sign of a growth showed itself. Professor W. H. Welch, Dr. S. Flexner, and myself each examined cover slips made from the vitreous body and anterior chamber, but discovered no organisms. I then obtained a rabbit and made a small opening into the anterior chamber with an iridectomy knife. The instruments were, of course, thoroughly sterilized. An iris forceps was introduced into the

FIG. 4.



Meridional section of the ciliary region in an eye which gave rise to sympathetic ophthalmia (reported on the preceding page). 1, conjunctiva thickened; 2, cornea showing small areas of leucocytes, which are especially marked near the ciliary region and increase in number towards the point of traumatism; 3, cyclitic membrane inflamed, fibrous, and thickly infiltrated with round cells; 4, ciliary body enlarged or densely infiltrated with round cells, especially towards angle.

anterior chamber of the enucleated eye and a regular iridectomy was performed. I found the iris very hard to pull away from its adhesions to the lens-capsule, but managed to draw out and cut off a good-sized piece of the iris. This latter was then forced into the anterior chamber of the rabbit's eye and moved about several times in the chamber and partly drawn out and left. In a week's time, with the exception of a slight blush about the wound, the rabbit's eye showed no evidence of disease, and in two weeks this injection had faded entirely away, and only a slight prolapse of the iris at this point gave any evidence of the operation. The cornea remained clear throughout, and the prolapsed iris was simply the result of the manipu-

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lations, and in no way interfered with the functions of the eye,—just what we sometimes see in cataract operations without iridectomy. During the operation I took pains to bruise the tissues about the incision, thus imitating as far as possible the conditions peculiar to a penetrating wound of the eye. We have here an undoubted case of sympathetic inflammation, and if bacteria in the injured eye gave rise to this inflammation, it is reasonable to suppose that the means employed to discover them would have been successful, and that the experiments on the rabbits would at least have suggested something which might be regarded as positive evidence.

It is interesting to note in this case the fact that, notwithstanding the violent inflammation which was present in the ciliary region, and which gave rise to the sympathetic ophthalmia, the posterior part of this eye was absolutely normal. It seems, then, that a traumatic irido-cyclitis may exist, and possibly run its course, without implicating the optic nerve, or even the retina and the chorioid, for any considerable distance posterior to the ora serrata, and it is doubly interesting to note that sympathetic ophthalmia can result in such a case. It is evident that the optic nerve played no part here in the sympathetic disease.

Not long since I met with a case in which a woman had been struck in the eye a year previously and sympathetic iritis serosa had developed two months afterwards. The injured eye was enucleated and subjected to the most careful bacteriological examination, but no evidence of the presence of organisms could be seen.

These two cases are the only ones of sympathetic ophthalmia in which I made a bacteriological examination. I have, however, made numerous such examinations of eyes enucleated for fear of an outbreak of the disease, and in three cases I have succeeded in demonstrating the presence of organisms at periods ranging from three to twenty days after the injury. One was a case of violent panophthalmitis resulting from the entrance into the eyeball of a particle of steel. The case was seen a week after the injury, and I succeeded in isolating the staphylococcus aureus of Rosenbach[1] in pure culture. The second was a case of panophthalmitis, in which I found the bacillus coli communis, the eye being enucleated on the fifth day after the reception of the injury. The third was a case of traumatic irido-cyclitis, in which the eye was removed on the twentieth day, and I was able to demonstrate the presence of the staphylococcus albus. The twentieth day, then, represents the latest stage of a traumatic irido-cyclitis in which I have succeeded in finding organisms; but of course no conclusions can be drawn from so small a number of observations.

It is reasonable to suppose that the sooner an eye is examined after the injury the greater the chances of finding organisms, and no doubt if all eyes affected with traumatic irido-cyclitis were removed within a few days of the

[1] Randolph, A Case of Panophthalmitis caused by the Bacillus Coli Communis, American Journal of the Medical Sciences, October, 1893.

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injury and an examination made for organisms, the latter would be found in every case. It is almost certain that such wounds are infected wounds; if not infected by the foreign body, they are rendered so by the entrance of organisms through the passage made by the former. Leber's,[1] Knapp's,[2] and my own experiments show that aseptic wounds cause little or no disturbance, and hence it seems perfectly clear that a penetrating wound of the eyeball is dangerous only when the wound becomes infected. When I say dangerous, I mean capable of giving rise to sympathetic inflammation. In nearly all such eyes a bacterial origin for the inflammation is demonstrable. I may mention here, as I have done elsewhere, that simply mechanical injuries of the ciliary nerves do not give rise to inflammation in the other eye. Take, for instance, glaucoma: the ciliary irritation sometimes is of so violent a type that not only is its reflex influence seen in the most excruciating ciliary neuralgia, but even in parts as remote as the stomach there is functional disturbance, manifesting itself in nausea and vomiting; and yet there is never any suggestion of sympathetic inflammation. Even in those cases where portions of the iris have been incarcerated in the corneo-scleral wound, and, as a consequence, the ciliary nerves put on a stretch, we do not see sympathetic inflammation make its appearance, unless, as Schirmer[3] remarks, an infectious irido-cyclitis exists.

Wounds of the ciliary region have always been thought to predispose peculiarly to sympathetic ophthalmia, and in this connection I[4] have performed quite a number of experiments on dogs. In every case the wound was made in the ciliary region with a sterilized instrument; prolapse of the iris was produced purposely in every case, as this kind of wound is thought by some to favor the development of sympathetic ophthalmia. In none of the cases did any excessive inflammatory phenomena follow in the operated eye, and there was never any suggestion of trouble in the other eye. Hyperæmia of the conjunctiva and iris in the vicinity of the wound was the only result. Two weeks later, on examination with the ophthalmoscope, I found the media perfectly clear and the fundus normal. The iris had healed in the corneo-scleral wound, there was no sensitiveness to touch, nor was there photophobia. The dogs were kept under observation for many weeks, but no pathological changes were ever observed.

Such results, I think, are instructive, for they at least show that in the case of the dog, just as in the case of man, injuries to the ciliary region are not in themselves sufficient to cause sympathetic ophthalmia, but that something else is necessary, a something modifying the character of the wound itself. If the ciliary nerves are concerned in the production of this disease, we must assume that the bacterial inflammation exercises a specific irritation on the nerve-endings, an irritation which affects these nerve-endings differently from a mechanical injury, and which is conveyed to the ganglion, and

[1] Leber, Archiv für Ophthalmologie, Bd. xxviii., ii.
[2] Knapp, Archives of Ophthalmology, vol. xv.
[3] O. Schirmer, loc. cit.
[4] Randolph, loc. cit.

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from that point passes over to the nerves of the other eyeball. This explanation, however, is not warranted by analogy with the history of any other bacterial inflammation of which we know, and I think is hardly admissible.

Schmidt-Rimpler[1] proposes, however, a modified ciliary-nerve theory. He bases his theory on the following two cases which he reported at the Heidelberg Congress:[2]

"In May, 1887, a man received a perforating wound of the sclera, involving the ciliary region. The wound was closed with a stitch. Healing at first went well, but two months later pain on pressure was felt, and phthisis bulbi set in. Enucleation was refused. Three months after this he came to me, and I found great tenderness on pressure and beginning phthisis bulbi. Sublimate injections were employed, and the tenderness on pressure disappeared, but irritability remained. On November 10 optico-ciliary neurotomy was performed, and one and one-half centimetres of the optic nerve were resected. Four days later the patient was discharged. The other eye was sound. Nearly two years afterwards he made his appearance with an affection of the other eye, that showed itself in intense pericorneal injection, iritis with posterior synechiæ, and clouding of the aqueous humor, and in addition to this it was not possible to see the fundus of the eye; V = 1/10. The primarily affected eye was tender on pressure, and, inasmuch as no improvement followed either a course of sweating or inunction, this eye was enucleated. The symptoms of irritation in the other eye disappeared, and the patient left with V = 5/10. Microscopical examination of the optic nerve stump and of the ball for organisms proved negative. The posterior ciliary nerves as well as the optic nerve were atrophic, while in the anterior portion of the ciliary body and in the cornea normal nerve-fibres were found. We have here a case of sympathetic ophthalmia in which bacteria have played no part.

"The other case was one in which sensitiveness to pressure in the primarily affected eye was wanting. In the second eye there was neuritis, with opacities in the vitreous body. The eye externally was pale and the pupil responsive to light. About this time the boy received a violent blow on the primarily affected eye, and the next day the sympathizing eye was more intensely injected and irritated, the pupil was narrower, and there was more photophobia. In four days both eyes were free of irritation."

It is not probable, Schmidt-Rimpler thinks, that bacteria had anything to do with causing the disease, inasmuch as the papilla of the left eye was normal, or very nearly so, whilst pronounced changes were present in the anterior part of the right eye. The fact that the process increased in intensity after the blow on the left eye would rather suggest a case of ciliary neurosis. These two cases cannot be explained by the infection theory, but may in fact be said to contradict that theory. He does not think the

[1] Schmidt-Rimpler, Beitrag zur Aetiologie und Prophylaxe der sympathischen Ophthalmie, Archiv für Ophthalmologie, Bd. xxxviii., 1892.

[2] Schmidt-Rimpler, Bericht der ophthalmolog. Gesellschaft, Heidelberg, 1891, S. 100.

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ciliary-nerve theory alone can explain the phenomena of sympathetic inflammation, though he seems to believe that many of the tissue-changes here are analogous to those brought about at other points by the action of vaso-motor or trophic nerves.

Were Schmidt-Rimpler's theory the right one, symptoms of sympathetic irritation would always usher in an attack of sympathetic inflammation, or at least be present during the outbreak of the disease in the other eye. It is well known that either of these conditions may be, and frequently is, absent, that sympathetic inflammation often makes its appearance without any symptoms of irritation, and that sympathetic irritation can exist for years without inflammation. In this connection, Theobald,[1] who is a strong advocate of the ciliary-nerve theory, is of the opinion that the ciliary nerves contain sympathetic as well as sensory fibres, and that it is likely that the former have most to do with trophic changes, the latter with disturbances of sensation. In view, then, of the great difference in the degree of pain inflicted in different persons, he thinks that in the eye, as well as in other parts of the body, these two kinds of fibres do not always exist in the same relative proportion, or at least that in different individuals sometimes the influence of one set and sometimes the influence of the other may predominate. This being the case, it is easy to understand why a given lesion of the ciliary body should give rise in one instance to pronounced sympathetic irritation unaccompanied by structural changes, and in another to trophic changes in the sympathizing eye with little or no accompanying irritation.

The following case, reported by Deutschmann,[2] is of more than usual interest because the man died of carcinoma of the stomach. The changes found in the sympathizing eye, in the opinion of Deutschmann, must have been the result of a process that necessarily was limited to the visual apparatus from beginning to end.

A man thirty years of age had undergone an unsuccessful operation upon his right eye six months before. Inflammation associated with great pain followed the operation, and sight was ultimately lost. The patient, when first seen, presented the following conditions. In the right eye there were phthisis bulbi and occlusion of the pupil. The cornea was clear, though there was a scar at the limbus marking the point where the incision was made at the time of operation. Slight ciliary injection was also present, and the eye was soft and extremely sensitive to pressure. The other eye was sound so far as external appearance and vision were concerned. The ophthalmoscope, however, showed a red papilla with indistinct edges, the vessels being dilated and the surrounding retina cloudy. The patient refused absolutely to submit to an operation, so he was put through a course of inunction, sweating, and iodide of potassium. It may be well to add that

[1] Theobald, Some Recent Theories regarding the Pathogeny of Sympathetic Ophthalmia viewed from a Macroscopic Stand-Point, Archives of Ophthalmology, xiii., 1884.

[2] Deutschmann, Beiträge zur Augenheilkunde, Bd. i.

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there was no evidence of syphilis, though the man was very pale and had a cachectic appearance. He had no fever, and complained of nothing in particular, except trouble in his stomach. The treatment with the above-mentioned agents proved fruitless. The primarily affected eye grew less irritable and gradually lost its sensitiveness to pressure, while the neuro-retinitis in the other eye increased and vision soon began to fail, followed by the appearance of fine opacities in the vitreous body. The patient was lost sight of for nearly three months, and when next seen the vision in his good eye was 1/15, and there were present pronounced neuro-retinitis, opacities in the vitreous body, and beginning ciliary injection. The other eye showed no further changes. The cachectic condition of the patient seemed worse, and he presented the appearance of one affected with carcinoma. He still refused to allow an enucleation, so he was put under active treatment with a view of bettering the condition of the left eye,—the eye in which sympathetic ophthalmia was present. Things continued to go from bad to worse; posterior synechiæ appeared, and the vitreous body became so cloudy that it was not possible to see the fundus. The vision sank to recognizing the movements of the hand. The general condition of the patient grew rapidly worse, till there was no longer any doubt about the existence of a carcinoma of the stomach. He died a few days later.

An autopsy was obtained, and both orbits, the eyeballs, the optic nerves, and the chiasm were secured for examination. It may be mentioned that at the autopsy the meninges showed nothing abnormal. The right eye was slightly atrophic, the optic nerve entrance decidedly spread out and infiltrated throughout with round cells. The trunks of the ciliary nerves at this point presented nothing abnormal; sometimes here and there in the sheath a round cell would be noticed. The tissue of the optic nerve itself was abundantly infiltrated with round cells, especially in the pial septa. The walls of the blood-vessels in the papilla were richly infiltrated with round cells. The retina, especially in the nerve-fibre layer, was markedly infiltrated, and at points was separated from the chorioid by an exudate. The vessels of the retina participated to the same extent in the process. The retina in the vicinity of the ora serrata did not show much infiltration, but was simply atrophic. The chorioid was atrophic very generally, and the pigment epithelium at points was detached and partly atrophied. Both the retinal infiltration and the chorioidal infiltration were less pronounced near the ciliary region. The vitreous was detached anteriorly and posteriorly and transformed into a fine fibrous tissue. The sclera was slightly infiltrated at the posterior pole. Infiltration of the sclera, as well as of the subconjunctival tissue, was noticed at the limbus of the cornea. The cornea was also infiltrated, and at its upper border was to be seen a scar, along the course of which cell-infiltration was very noticeable. The iris and ciliary body showed atrophy of the pigment elements. Posterior synechiæ were present, the synechiæ consisting of nucleated fibrous tissue containing round cells, particles

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of pigment, and endothelioid cells, forming a tissue which completely closed the pupillary opening. The anterior chamber contained an exudate that consisted of round cells and threads of fibrin. Especially noteworthy was an anterior synechia unassociated with a corneal scar. There was hardly more than a rudiment of the lens remaining.

As regards the condition of the second eye, the optic nerve, at the point where it passed into the eyeball, was richly infiltrated with round cells, and this was particularly noticeable in the pial septa. The ciliary nerves at this point were intact, except that here and there a round cell could be noticed. The papilla was very much swollen and infiltrated with round cells, and the sheaths of the central vessels participated in this process. The retina, especially in the fibre layer and as far forward as the ora serrata, was the seat of round-cell infiltration, the infiltration showing even to some extent in the ora serrata. The chorioid was richly infiltrated, the cells sometimes occurring in little groups or heaps, and sometimes being uniformly distributed throughout the tissue. The pigment epithelium was loosened. The round-cell infiltration of the chorioid appeared to be less pronounced a short distance forward from the equator, but beyond this up to the ciliary body and in the latter the cell-infiltration was abundant. The sclera in general was intact, though the walls of the small blood-vessels penetrating the sclera were infiltrated. The vitreous body was somewhat shrivelled, and at some points was detached from the retina. Throughout the vitreous body were to be seen numerous cells, partly small round cells, partly large cells with vacuoles and containing several nuclei. The posterior chamber contained coagulated fibrin mixed with round cells. The lens was intact, except that there was an abundant pigment deposit on the anterior capsule, and in addition to this an exudate that soldered the anterior capsule to the posterior surface of the lens. The ciliary body was densely infiltrated with round cells. The anterior chamber contained a fibrinous exudate rich in cells. The iris was infiltrated throughout with round cells, and the cornea showed a slight infiltration at the scleral border, as did also the subconjunctival tissue at this point. The external sheath of the optic nerve of the primarily affected eye all the way up to the chiasm was only moderately affected. The inner sheath, however, was considerably infiltrated with round cells, the infiltration being here and there quite dense, and at other points more diffuse. The cell-infiltration was pronounced in the pial septa. At the chiasm the inner sheath of the nerve was markedly infiltrated. The pia mater was infiltrated only in the immediate vicinity of the chiasm; otherwise it was perfectly normal. The other optic nerve showed also pronounced round-cell infiltration of the inner sheath at the chiasm. This cell-infiltration continued with variable intensity all the way down to the second eye. Both orbits were normal, and there was here no suggestion of involvement on the part of nerves, muscles, or blood-vessels. The ciliary nerves, wherever met with in the sections, were perfectly normal. The brain and its mem-

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branes were normal. Micro-organisms were found in both eyes, and also in the optic nerves. The organisms had somewhat the appearance of gonococci, and in the primarily affected eye were observed to be most numerous in the ciliary body and iris and chorioid, especially in that part of the chorioid near the papilla. They were also to be seen in the retina, in the papilla, and about the central vessels. In the optic nerve organisms were found in the nerve-trunk and in the walls of the smaller blood-vessels which pass through the inner sheath of the nerve. No organisms were found in the outer sheath of the nerve. The condition was the same in both optic nerves and in the chiasm. Some few organisms were found in the pia mater in the immediate vicinity of the chiasm. In the second eye micro-organisms were found to be most abundant in the posterior part of the eye, just as was the case with the first eye. There were no organisms in the orbits. It will be remembered that neuritis optica existed for some time in the second eye before there was disturbance of vision.

Deutschmann thought that the neuritis was produced by the chemical and metabolic products that preceded the migration of the organisms, and that it was only where the latter had traversed the optic nerves and passed into the second eye that the disease spread with rapidity and with its usual destructiveness. He regarded the optic nerves and chiasm as the route followed by the organisms, and believed that the pial sheath of the nerve and the nerve-trunk were the parts preferably attacked; and he concluded by stating his position again with respect to the pathogenesis of sympathetic ophthalmia, a position which is well known.

Such a case is of undoubted value, for if a general infection can be absolutely excluded we are not far from the solution of this problem. The observation, however, is an isolated one, and, while we cannot but appreciate its value, it will never be looked upon as conclusive, so long as the experimental side of the question remains so one-sided. It may be added that a general infection cannot possibly be excluded without a bacteriological examination of the blood and other organs, as such general infection, especially with streptococci, is not infrequent as a terminal event in various chronic diseases, including cancer of the stomach. The many negative results do not disprove the bacteric origin of sympathetic ophthalmia, but before regarding such a theory as proved the specific organism must be identified, and especially should this be the case with an affection like sympathetic ophthalmia, a disease the pathogenesis of which really does admit of more than one reasonable interpretation.

PROGNOSIS.

Under any circumstances the prognosis is a matter of doubt. Well-established recoveries are rare. Secondi [1] reports a case, and Waldispuhl,[2] in summing up the statistics of Professor Schiess's clinic in Bâle, reports

[1] Secondi, Centralblatt für praktische Augenheilkunde, July, 1892, S. 224.
[2] Waldispuhl, Inaug. Diss., Luzerne, 1892.

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four recoveries in ten years. Cases of recovery are reported by Hirschberg,[1] Laqueur,[2] Schirmer,[3] and Rogman.[4] Including all these cases, I have been able to collect nineteen well-established recoveries from sympathetic plastic uveitis which have occurred during the past twenty years. Laqueur thinks that the age of the patient is of importance in the successful termination of the disease. If the patient is young, the chances for recovery are better, because the high degree of nutrition of the tissues in youth is able to triumph over the serious obstacles produced by the disease. Most of the cases which have recovered were under forty years of age, and many of them were children. The following case is one which I saw early in the winter of 1895. It is especially interesting as affording an opportunity of studying the changes in the fundus, and these changes have been indicated in the accompanying water-color.

G. W. came to the Johns Hopkins Hospital for treatment. He had been struck in the left eye by a piece of stone, and when first seen, four days later, had a small penetrating wound of the eyeball at the upper and inner border of the cornea. The pupillary area was nearly filled with a filmy exudate, and the pupil was contracted. There was a faint line of what appeared to be pus at the bottom of the anterior chamber. He could count fingers at six feet. The other eye was normal in every respect; and I may say here that he had never had any previous trouble with his eyes. There was no history of any constitutional trouble. The injured eye was very much congested and sensitive to the touch. A compress bandage was applied, atropine was ordered to be used every three hours, and he was given small doses of calomel. Three days later the condition had become much worse. The anterior chamber was more than half full of pus, and only light-perception was present. The anterior chamber was emptied by a paracentesis, and, rather to my surprise, there was no reformation of the pus. The pupil yielded gradually to the mydriatic, and full dilatation was obtained. At the end of the fifth week he could count fingers at fifteen feet. The eye was still congested, the vitreous body hazy, and there was sensitiveness to the touch. He left the clinic, and returned in four days with the report that he could hardly see anything with his good eye. On examining the right eye there were found two adhesions at the lower and temporal border of the pupil and another at the inner and upper border. The vitreous was so cloudy that it was impossible to get any satisfactory view of the fundus. He had suffered no pain. There was considerable circumcorneal injection. It was a typical picture of plastic uveitis. His vision in this eye was 16/200. Here, then, was a case of sympathetic ophthalmia with useful vision still present in the injured eye, the vision in the latter still being the ability to count fingers at fifteen feet. This condition of affairs continued

[1] Hirschberg, Centralblatt für praktische Augenheilkunde, Oct. 1891, and March, 1895.
[2] Laqueur, Annales d'Oculistique, Nov. 1895.
[3] O. Schirmer, loc. cit.
[4] Rogman, Annales d'Oculistique, August, 1895.

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till the beginning of the ninth week, when for no apparent reason vision in the injured eye sank to light-perception, and in two days light-perception had disappeared. These changes were associated with marked decrease in intra-ocular tension. The necessity for retaining the eye no longer existed, so it was enucleated, on the ground that, as it was the focus of the sympathetic trouble, it might still be exerting a pernicious influence upon the other eye. Soon after this the sympathetically affected eye began to improve. During all this time the man was taking one-sixteenth gr. hydrarg. bichlor. and ten gr. potass. iod. three times a day, and at least once a week his bowels were thoroughly moved by small doses of calomel. His vision at the end of the ninth week was 16/200, and now, in the fourteenth month, his vision is 16/40, and he can make out two or three letters on the next lower line. There is a slight adhesion at the upper and inner border of the pupil, but the latter is, with this exception, symmetrically dilated. There are several small pigment-spots on the anterior capsule. The media are perfectly clear, and only recently has it been possible to see the fundus. Relapses have been known to occur at a later period than a year, so that this patient cannot be pronounced absolutely out of danger. We may say, however, that, as a rule, when one has passed through a year without a relapse the conditions justify a favorable prognosis. Under a year one should be reserved in making any statements as to complete recovery.

TREATMENT.

The prophylactic treatment naturally plays a most important rôle in dealing with sympathetic ophthalmia, and of course the only certain prophylaxis is the enucleation of the injured eye. Wardrop in 1819 proposed emptying the contents of the eyeball by a large incision through the middle of the eye. Enucleation was first practised by Prichard, though the credit of having suggested such a step is given by some to von Ammon, by others to White Cooper. By certain prophylaxis I mean relatively certain, for we can never assert positively that sympathetic ophthalmia will be surely averted if the injured eye be enucleated, nor, indeed, can the assertion be made that in any case where the other eye is perfectly sound it will become diseased unless the injured eye be enucleated.

Mooren[1] and Schmidt-Rimpler[2] each report a case in which at the enucleation the other eye was perfectly sound, yet sympathetic ophthalmia broke out in one case on the fourth day and in the other on the second day. Again, a case is reported in which blindness followed, and the sympathetic inflammation did not show itself till nine days after the injured eye was removed. As a general thing, in the many cases of this kind that have been reported the sympathetic disease showed itself within two or three weeks

[1] Mooren, Ueber sympathische Gesichtsstörungen, Berlin, 1869.

[2] Schmidt-Rimpler, Sympathische Ophthalmie, Klinische Monatsblatt f. Augenheilkunde, xii., 1874.

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after the enucleation. Cross[1] reports a case in which the inflammation appeared twenty-six days after the enucleation of the injured eye. Nettleship[2] reports three cases occurring in twenty-two, twenty-three, and twenty-four days; and Snell[3] mentions a case where thirty-two days after the first eye was removed sympathetic ophthalmia broke out in the other eye. In Nettleship's[4] Committee Report two cases are recorded in which the injury had resulted in rupture of the eyeball, followed by violent inflammation of the orbital tissue. The inflammation did not subside after the enucleation of the lacerated eyeball, and in one case sympathetic disease showed itself in the other eye seven weeks later, and in the other case five weeks later. Brailey's[5] explanation is that infection took place from the orbital tissue.

Evisceration[6] was introduced by Alfred Graefe in 1884, and later was taken up by Mules. It was asserted that it was a far less dangerous operation and furnished a better stump for an artificial eye. As to its being a less dangerous operation I am very doubtful, for Schulek,[7] of Budapest, reports a case of death after this operation, and Cross[8] reports two cases of sympathetic inflammation in the other eye three weeks later. Hotz[9] observed a case of sympathetic neuritis. In Graefe's cases the average time for recovery was nine days. Frequently great pain that has persisted for some days has been experienced after this operation, this being a feature but exceptionally seen after enucleation. As regards the motility of such a stump, Hotz[9] has demonstrated that patients, when evisceration was performed, possessed no greater power of rotating the artificial eye inward and outward than after enucleation. The operation seems, then, of very doubtful value, and I am not disposed to recognize any condition in which enucleation would not be preferable.

The same may be said of optico-ciliary neurotomy, first practised by Boucheron.[10] Redard's[11] experiments on rabbits and dogs have shown that the cut ends of the optic nerve do grow together perfectly, and Mauthner[12] reports a case where the nerve-ends grew together in a human being. Of course in these cases there is permanent loss of function. But when we come to speak of the ciliary nerves we find a different state of affairs. Here in most cases a partial restoration of function is almost invariable, and some cases are reported in which function was completely

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[1] Cross, Ophthalmic Review, 1887, p. 236, Case 3.

[2] Nettleship, Transactions of the Clinical Society of London, vol. viii., 1880.

[3] Snell, Transactions of the Oph. Soc. of the United Kingdom, 1882.

[4] Nettleship, Committee Report on Two Hundred Cases of Sympathetic Ophthalmia.

[5] Brailey, On Sympathetic Ophthalmia, Tenth Inter. Med. Congress in Berlin, 1890.

[6] This operation was performed by Noyes in 1872 (Diseases of the Eye, 2d edition, p. 401), and by Williams, of Boston, in 1877 (Trans. Am. Ophth. Soc., 1878, p. 406).

[7] Schulek, quoted in Fuchs's Diseases of the Eye, p. 322.

[8] Cross, Sympathetic Ophthalmitis after Evisceration, Ophthalmic Review, 1887, p. 236.

[9] Hotz, Jour. Amer. Med. Assoc., October 21, 1893.

[10] Boucheron, Névrotomie optico-ciliaire, Gazette des Hôpitaux, Paris, 1890.

[11] Redard, Recueil d'Opht., 1880.

[12] Mauthner, loc. cit.

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regained. The case reported by Mauthner was one in which the operation of neurotomy had entirely removed the symptoms of sympathetic irritation. Sensibility, however, returned to the operated eye, and all the symptoms of irritation reappeared in the fellow-eye, and enucleation had to be performed. A somewhat similar case is reported by Poncet.[1]

Leber[2] reports a case of genuine sympathetic inflammation after neurotomy. The case was one of plastic irido-cyclitis of traumatic origin. Three months after the injury neurotomy was performed, and ten days after this the eyeball was absolutely insensitive. In two years, however, he returned with sympathetic iritis, while the injured eye was sensitive to the touch, a condition that had been present for several months before the outbreak of the sympathetic disease. The eye in which neurotomy had been performed was then enucleated, and it was found that connection had been re-established between the cut ends of the nerve. The operation is not infrequently performed by Chisolm in cases of absolute glaucoma associated with persistent pain, simply to preserve the eyeball; but in these cases more than once it has been found necessary to enucleate the eye on account of the return of the pain. As a means, then, of warding off sympathetic ophthalmia, I regard neurotomy as utterly uncertain, and consequently taking a far lower rank than enucleation, evisceration, or resection of the optic nerve.

This latter operation—a modification of optico-ciliary neurotomy—is strongly recommended by Schweigger,[3] and holds out a better prospect for safety than neurotomy. Scheffel[4] has collected forty-one cases in which resection of the optic nerve was performed, and good results followed in every case where the sympathetic affection was of the irritative type. The following two cases of sympathetic ophthalmia are reported by Clausen[5] and Ohlemann. In both cases resection of the optic nerve of the injured eye had been performed. In the first instance the patient had been wounded in the right eye with a pair of scissors nine years before, and during all this time had experienced no trouble with the eye. Sight, of course, had been long extinguished, and the eye was undergoing phthisis. Without any external cause, the eye became injected and very sensitive to the touch. Resection was performed. Seventeen days later symptoms of sympathetic disease appeared in the fellow-eye, and permanent impairment of vision resulted.

Ohlemann's[6] case was as follows. A piece of steel had been removed

[1] Poncet, Discussion on Sympathetic Ophthalmia, Trans. Int. Med. Cong., Lond., 1881.

[2] Leber, Bemerkungen über die Entstehung der sympathischen Augenerkrankungen, Archiv für Ophthalmologie, 1881, xxvii. 331.

[3] Schweigger, Augenheilkunde, p. 337.

[4] Scheffel, Ueber Sehnerven-Resection, Klin. Monatsbl. für Augenheilk., 1890.

[5] Clausen, Ein Fall von sympathischer Ophthalmie trotz Resection des Opticus, Diss. Inaug., Kiel, 1886.

[6] Ohlemann, Die perforirenden Augenverletzungen mit Rücksicht auf das Vorkommen der sympathischen Ophthalmie, Archiv für Augenheilk., xxii., 1891.

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from the interior of the eyeball with a magnet, and at first recovery went along smoothly, but two weeks later the injection of the eye became very intense and the eyeball sensitive to the touch, and in consequence of these symptoms resection of the optic nerve was performed. In ten weeks the operated eye had regained its sensibility and iritis serosa had broken out in the other eye. Trousseau[1] and Schmidt-Rimpler[2] both report cases of a similar nature. It seems quite evident, then, that not even the cutting out of a piece of the optic nerve is sufficient to prevent sympathetic ophthalmia. This fact would seem to throw a doubt upon the theory that the optic nerve is the channel of communication, though there is only one case, that of Schmidt-Rimpler, in which, after the removal of fifteen millimetres of the optic nerve, sympathetic disease broke out. There is no doubt, however, that resection does render difficult an outbreak of sympathetic ophthalmia, and is a much more reliable procedure than either evisceration or neurotomy.

In those cases in which sympathetic irritation exists, and we have no special reason to believe that sympathetic inflammation will appear, I think we may safely employ resection; and this applies especially to those eyes that have become blind from other causes than penetrating wounds, for instance, in which absolute glaucoma exists, or in those cases where an inflammation has destroyed the cornea and phthisis bulbi follows. In case an eye is blind, however, from a penetrating wound, the safest course to pursue would be to enucleate. An eye that is capable of giving rise to sympathetic ophthalmia is a dangerous eye, and should be removed without waiting for symptoms of irritation in the other eye.

In those cases where the first eye has some vision or a possibility of it, it is a very difficult question to decide what to do. One has to determine whether the vision in the injured eye is good enough to warrant the hope of an improvement, and this conclusion can be reached only by carefully studying the nature of the wound and considering what the outlook generally is in wounds of a similar character. And, in spite of the greatest care and conscientiousness, one will make mistakes sometimes in deciding upon what course to follow in this class of cases. Here there is no invariable law to follow. I think the best guides in a case of this kind are the tension and the sensitiveness to touch. If the latter condition is marked and the tension decidedly lowered, and at the same time only light-perception is present, the chances of improvement for this eye are bad, and especially so if these conditions persist for three weeks after the injury; and in such a case I think it would be running a risk to delay enucleation.

When the injured eye is blind and sympathetic irritation is present in the other eye, it is wise to enucleate.

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[1] Trousseau, Un cas d'Ophthalmie sympathique malgré la Resection du Nerf optique, Société d'Opht. de Paris, Séance de 7 Avril, 1891.

[2] Schmidt-Rimpler, Beitrag zur Entstehung der sympathischen Ophthalmie und Discussion, Heidelberger Congress, 1891.

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In case the first eye possesses a little vision, and symptoms of irritation are present in the second eye, every effort must be made to improve the condition of the first eye, and this means to apply the rules governing the treatment of any irido-cyclitis, and, if necessary, the performance of iridectomy.

When sympathetic inflammation has already broken out, I should remove the injured eye if it is blind. Cases are reported by von Graefe,[1] Power,[2] Bresgen,[3] Samelsohn,[4] and others in which recovery followed without the removal of the injured eye. If the injured eye is not blind, the same course should be pursued as is suggested when the condition is that of sympathetic irritation in the second eye: in other words, do not enucleate.

Hirschberg[5] reports a case of sympathetic ophthalmia in which the conditions were of this character, and in which the second eye was lost and the first continued to have sight. The following interesting case is reported by Schirmer.[6] A boy, eight years old, was struck in the right eye. Three weeks later the iris was hyperæmic, the anterior chamber shallow, the cornea cloudy, and the eye very much injected. There was quantitative light-perception, and tension was minus. There was no sensitiveness to pressure. The left eye was normal. Less than ten days later pericorneal injection appeared in the left eye, associated with deep-seated cloudiness of the cornea and deposits on Descemet's membrane. The vitreous body and the fundus were normal. He was put to bed, and atropine was employed locally and salicylate of sodium internally, with the effect of clearing up in a large measure the corneal cloudiness. This treatment was kept up for several months, and he was finally discharged. Eight months after the injury the eye was free from irritation. The corneal clouding in the right eye had cleared up to such an extent that fingers could be counted at twenty-five centimetres. In the left eye vision was over 2/3.

Mauthner[7] advises against enucleation where serous iritis only is present in the second eye, giving as his opinion that such an iritis may be transformed into a malignant iritis by the operation. He supports his views by references to three cases reported by Knapp, Derby, and Mooren. His arguments, however, are far from convincing, and, indeed, there is no clear reason for thinking that such a termination was directly traceable to the operation.

As to medicinal agents, we possess nothing that seems to exercise a specifically beneficial influence in the treatment of sympathetic ophthalmia. In the early stages we may use atropine guardedly, but this must not be

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[1] v. Graefe, Archiv für Ophthalmologie, xxii. 2.
[2] Power, Royal London Ophthalmic Hospital Reports, vol. vii., 1873.
[3] Bresgen, Wiener medizinische Wochenschrift, Nr. 45 u. 46, 1878.
[4] Samelsohn, Archives of Ophthalmology, vol. iv.
[5] Hirschberg, quoted by Noyes, Diseases of the Eye, p. 495.
[6] O. Schirmer, loc. cit.
[7] Mauthner, loc. cit.

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persisted in, for more harm than good may ensue. The eye should be absolutely at rest. Hot fomentations do good service, and they should be used several times daily, but I think the best way to apply heat is in the shape of dry heat. A most convenient form is that known as the "Japan stove" or "hot box." Darkness is essential, and a good tonic is strongly indicated.

Pain may be alleviated by the instillation of cocaine. There is a difference of opinion on the subject of mercurials, but calomel in small doses was certainly helpful in the cases in which I have seen it employed, and in the case reported by me in the foregoing pages I attribute the happy issue in large measure to the use of mercurials. Noyes [1] mentions a case in which the disease was arrested by inducing salivation. Of course we may apply the drug through inunction. Injections of pilocarpine have been known to do good; also the administration of the fluid extract of jaborandi. Operative interference must be put off as long as possible. The opening made by an iridectomy is sooner or later closed up with a fresh exudate.

Not a little has been written during the last three years of a method of treatment followed by Abadie.[2] The latter believes in the infectious nature of sympathetic ophthalmia, and is of the opinion that the injured eye is constantly supplying organisms that pass over to the healthy eye. His idea, then, is to disinfect the source of the disease, and this he proposes to do by the injection into the eyeball of a drop of sublimate solution 1 to 1000. At the same time he uses the galvano-cautery at the point of the injury; in other words, where the infection entered. He reports three cases, and in two of these, by the help of this method, he brought the sympathetic ophthalmia to a stand-still. The third case is especially interesting because the sympathetic ophthalmia persisted in spite of the enucleation of the injured eye, and yet by the injection of a drop of a 1 to 1000 sublimate solution the disease was arrested. This would certainly seem a valuable method of treatment, and it is rather surprising that it has not been more widely applied. But few observations, however, of a similar character from others are recorded. The treatment is worthy of a more extended trial.

The influence of an operation seems to be invariably a hurtful one. The following instructive case is reported by Schirmer.[3] A boy, nineteen years of age, was suffering with gonorrhœal ophthalmia. The inflammation ran a very mild course in his left eye and did not attack the cornea. The other eye was destroyed and was left with an incarcerated iris and an opaque cornea. Six months after he was infected his right eye was enucleated, and eighteen days after the enucleation irido-cyclitis broke out in the other eye. There were intense ciliary injection, epiphora, and photo-

[1] Noyes, Diseases of the Eye, 1890, p. 490.

[2] Abadie, Pathogénie et nouveau traitement de l'Ophtalmie sympathique, Annales d'Oculistique, t. ciii. p. 183.

[3] O. Schirmer, loc. cit.

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phobia. The cornea was dull, the anterior chamber increased in depth, and the aqueous humor cloudy. The iris was markedly hyperæmic; the pupil was rigid and filled with an exudate, but could be dilated under atropine. The ciliary body was very sensitive to pressure. Tension elevated. Vision much lowered. Atropine, hot applications, and inunctions were ordered. Under this treatment the clouding gradually grew fainter, and after six weeks he could count fingers across the room. The pupil was adherent all around, and the ciliary border of the iris pushed forward. An iridectomy was then performed, and a wide coloboma was obtained, which immediately became blocked up with blood. Inunctions were continued, but with no effect, and resort was made to another iridectomy. Only small particles of the iris could be removed. There was considerable hemorrhage, and no improvement of vision followed; in fact, the condition of the eye grew rapidly worse, till finally light-perception disappeared. Five other equally instructive cases are reported by Schirmer, and all go to show the unfortunate consequences attending an operation upon this class of cases. Operative interference with cases of this kind is justifiable only when the pain becomes unbearable, and here sclerotomy may do good by relieving the condition of secondary glaucoma which is frequently present. Lawson reports favorable results after this operation.

It seems, then, that sympathetic ophthalmia may terminate first and very rarely in recovery, and, on the other hand, that the eye may become phthisical and collapse.

There is still another termination, in which the eye retains its shape, but the iris is bound down by extensive adhesions to the lens, and the latter is opaque and only light-perception is present. What should be done in this class of cases? Here, as Critchett[1] says, the chief obstacle to vision is the lens. He suggests the following operation.

In the first place, every evidence of an acute inflammation must have passed away. A fine needle is directed to the centre of the opaque capsule, and the latter is pierced. Another needle is passed in from the opposite side, and, by bringing the penetrating force of one needle to bear upon the other, a small opening is made in the capsule. The points of the needles are then separated from each other. In this way quite a rent is made. There is generally an escape of lens-matter. Little or no reaction follows. An interval of several weeks is allowed to pass to permit of the absorption of lens-substance as far as possible, and then the operation is repeated, and so on, the operation being performed every time with two needles. Both Critchett and Story[2] report several cases in which useful vision resulted from this operation often repeated, and in which before the operation all hope of ever seeing again had been abandoned.

[1] Critchett, Royal London Ophth. Hosp. Reports, vol. x., Part 2.

[2] Story, Trans. Royal Acad. Med. Ireland, Dublin, 1890–91, ix. 422.

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OPERATIONS USUALLY PERFORMED IN EYE-SURGERY.

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I SHALL begin the description of the operations usually performed in eye-surgery with those that are specifically ophthalmological, the operations on the iris and the crystalline body, continue by those on the cornea, the conjunctiva, and the eyeball, then take up the operations performed on the ocular muscles, and conclude with the operative treatment of the diseases of the lacrymal apparatus, the orbit and its neighboring cavities, and the removal of foreign bodies from the eye. The operations on the eyelids are described by Dr. Harlan in another article.

§ I. GENERAL PREPARATIONS AND PRECAUTIONS.

It is self-evident that patients who have to undergo important eye-operations, especially those for cataract, should be in good physical condition. Yet it is an experience of late years that certain constitutional diseases—*e.g.*, albuminuria and diabetes—do not influence the recovery from a cataract extraction materially. If we choose a period when the patients are not particularly debilitated, we can operate on such patients with the average rate of success. It is advisable, however, not to let them lie on their backs for more than one or a few days, and to keep up their nutrition and strength, lest hypostatic pneumonia, pleurisy, and coma set in. This rule holds for old persons in general. There is no absolute contra-indication to letting a patient sit up from the day after the operation until his discharge.

Rheumatism and *gout* have an unfavorable influence on the recovery, on account of their predisposition towards iritic and cyclitic processes.

Drunkards ought to have a sufficient allowance of alcohol to preclude delirium tremens if possible.

An operation during acute *bronchitis* is unjustifiable, but *chronic bronchitis* need not be considered a contra-indication even to extraction. I have repeatedly seen persons that had prolonged attacks of violent coughing every night, recover from simple extraction without iris prolapse. *Very old and decrepit people may die soon after the operation*, some within a few days, others suddenly.

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I operated on an old woman who had been blind for many years. For years she had a great longing of seeing again, and expected from the restoration of her sight her return to youthful happiness. I operated on both her eyes by the flap extraction. The recovery was undisturbed. When from the fifth day on she was allowed to use her eyes she was disappointed. Her sight was excellent, but from day to day the more she used her eyes the more she felt depressed, and on the fifteenth day, when she was to leave the hospital, she died without a struggle, simply like a lamp the oil of which had been consumed. The expectation of regaining her sight seemed to be the only stimulus that kept her alive, and when that stimulus was no longer present she passed away.

Professor L. Laqueur, of Strassburg, mentions that he had observed five cases of *sudden death* between the fifth and twelfth days after an extraction. In all cases the autopsy discovered marasmic thrombosis of the veins of the legs and embolism of the pulmonary artery. Such experience should warn us not to operate on very old or decrepit persons without good reason. *Old age in itself is no contra-indication,* yet, taken all in all, it gives a worse prognosis than middle life.

Before performing an important operation on the eyeball, in particular cataract extraction, *all general and local septic affections ought to be excluded or cured.* Dacryocystoblennorrhœa is the most dangerous local complication; chronic muco-purulent conjunctivitis follows next; chronic trachoma, free from purulence, does not interfere seriously with primary union, nor does the acute conjunctival catarrh (pink-eye) or chronic dacryocystitis, if the contents of the lacrymal sac are watery or slightly viscid but not purulent. Yet all these conditions have to be closely examined. If the conjunctiva is smooth and simply red and swollen, its infectiousness is not great; but if it is dull, finely granular, like shagreen, it is not to be trusted. Bacteriological investigations have revealed pathogenic bacteria in every conjunctiva; practically we may be guided by the appearance and the secretion of the conjunctiva. Some operators have washed out the conjunctiva with antiseptic solutions several days before the operation, others keep the eye to be operated on bandaged for twenty-four hours, and base their judgment on the presence and quantity of the discharge noticed on removal of the dressing. Both the antiseptics and the dressings produce a certain degree of irritation. I am in the habit of keeping the patients, at home or in the hospital, in well-ventilated rooms, free from dust and tobacco-smoke, and regulate their general condition,—viz., the skin by a bath, the head and beard by shampooing, the bowels, if necessary, by an aperient suited to the individuality of the patient, and the eye by simply keeping it clean by washing it with soap and water morning and evening. If the eyes are free from discharge, and the conjunctivæ smooth and not congested, I consider the patients in good condition for the operation.

That the operator and everything coming in contact with the wound should be aseptic is self-evident.

It is of great importance *that the operator have good sight,* and that the *field of the operation be well illuminated.* The old rule, "*Sit chirurgus*

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juvenis," is particularly important in eye-surgery; the opacities in the refractive media and their surfaces of separation are often the objects of delicate surgical interference, which without good sight and good illumination could not be done with the necessary precision. Owing to the progress in the correction of refractive errors, an old operator may have as sharp sight as a young one. A presbyopic and astigmatic hyperope can, by double-focus spectacles, adjust his eye for any distance he likes, correcting through the upper part his refractive error, and through the lower make the visual object perfectly distinct, and even magnified, for the distance in which he is accustomed to operate. This is particularly important in operations on the capsule of the lens. The different magnifying contrivances recommended from time to time have, as far as I have tried them, not supplanted my convex glasses, nor have I seen them used by any other operator.

The patient may be operated at any hour of the day. If the weather be cloudy, a broad light from an Argand gas-burner, or the incandescent gas-light, or the electric light thrown upon the eye with an electrophore, gives an illumination which has all the advantages of daylight and some of its own. The room being darkened, there is no false light to interfere in recognizing the strongly illuminated minute objects of focal illumination. Operations of the capsule, for instance, could not be satisfactorily done by ordinary, nor even good, daylight. Most operations, extraction of cataract included, can be done by diffused daylight or by artificial light. Diffused light gives the operator greater freedom of manipulation, and, if need be, an assistant may always with a hand-lens throw the light from a window on the eye. Satisfactory arrangements for artificial illumination should be in every ophthalmic institute.

The operator, if possible, should be ambidextrous. He may perform correctly every eye-operation with the right hand, but ambidexterity offers many conveniences in position and the handling of instruments. It is not a gift of nature, but has to be learned. We should practise the left hand just as the pianist does, and when the first operations present themselves where the left hand should be used,—say, for instance, in an upward extraction on the left eye,—we should do it, and let no nervousness intervene, saying this time I rather use the right hand. This yielding will recur at the second and the third operation, and so on until the left hand is hopelessly given up. An upward section on the left eye can be made with the right hand, the operator sitting or standing before the patient; but the position is awkward. When the section is made, and iridectomy has to be done, the operator has to give the fixing forceps to an assistant, and has to dance around the patient's head in a semicircle, take the iris-forceps in the left hand, and cut the iris with the right. Moreover, as I have seen, if the operator cannot intrust the iris-forceps to his left hand, he must draw out a piece of iris with the right, and have it cut by an assistant.

If a left-handed surgeon be not ambidextrous he should have certain

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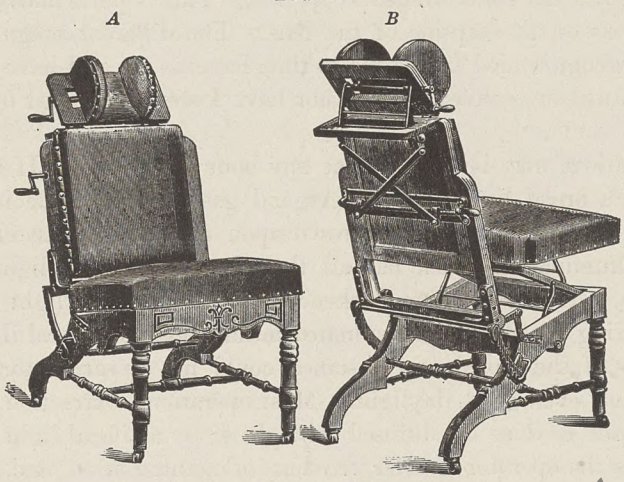
instruments—for instance, scissors—specially made for himself. If the scissors are very accurately made they may do for both hands, but if there is the least looseness in the lock the blades will separate, which makes their contact imperfect. This is particularly annoying when we have a tough membrane to cut, for instance, the tendon in a squint operation, or the cornea if an insufficient section is to be enlarged.

The patient is operated upon on an *operating chair or table.* The model represented by Fig. 1, A and B, is very convenient.

FIG. 1.



Operating chair.

It was first made in 1865, by the celebrated instrument-maker Fischer, of Heidelberg, who came to me and asked what requisites an eye-operating chair should have. When I had told him what I considered desirable he constructed the chair, which has become very popular both in Europe and America. It can be lowered and raised, and very easily turned on casters, so that the operator can find the best illumination and turn aside the disturbing corneal reflex. The head of the patient should be so placed that the operator's hands are at the level of the patient's eye when the forearm is slightly bent below the horizontal. This is the easiest position for sight and the least straining. If the patient's head is higher, the operator has to raise his forearm above the horizontal, which tires him and may make him shake.

For cataract extraction it is an advantage to operate on the patient in his bed, of which method the late Professor Arlt, of Vienna, was a great advocate. Perfect arrangements in this line exist in some modern institutions, for instance, in the new building of the New York Eye and Ear Infirmary. The beds can be moved on casters to the window or anywhere to the source of light; they can, without a shake, be rolled into the elevator and transported to the operating room and back again. When operated on an operating chair, which has the advantage of being handier than a bed, the patient, after the operation, has to be wheeled back and put to bed. For some years I have tried to make a virtue of necessity. I have operated on the patient in an operating chair, as I always do, and when I had made a

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simple extraction I put him to bed with his eyes unbandaged. I let him lie a quarter of an hour or longer with his eyes closed, then I inspected the operated eye again, and if the iris was still in position I bandaged both eyes, assuming that there was no tendency to prolapse. If the iris was displaced, I at once made an iridectomy. This happened five times in several hundred simple extractions. On longer trial I came to the conviction that all these special arrangements are of no essential importance.

Anæsthesia.—In the great majority of cases cocaine anæsthesia will suffice, but in children and nervous persons full general anæsthesia is necessary. This is also the case if we have to operate on a glaucomatous eye with high tension, where cocaine is inefficient, and on a prolapse of the iris after extraction, if the patient is afraid. Cocaine anæsthesia should not be too deep: two drops of a four per cent. solution, instilled three times, at an interval of four or five minutes, is sufficient. Deep cocainization renders the eye too soft, which, especially in old persons, makes the expulsion of the lens difficult and lets the cornea sink in like a funnel, which, however, is of no significance. For general anæsthesia I have, since 1871, constantly employed ether. I give ether at an average more than once a day. In short operations—most operations on the eye are short—I give it on the choking plan, which, timed for several hundred successive cases, took at an average one minute and thirty-seven seconds to be complete. On the other hand, in not a few cases I have kept patients under ether for one and several hours. In some cases the etherization was laborious from bronchitis, etc., but never have I had a fatal case, and only in a few cases had I to resort to artificial respiration. As long as I used chloroform, resuscitation manœuvres had to be made about once a month. I had no fatal case with chloroform either, but I never gave chloroform with the confidence with which I give ether. Finding ether satisfactory, I have not tried other anæsthetics. At the request of patients, I had to operate several times under laughing-gas anæsthesia, administered by a special anæsthetizer. It answered the purpose, even in longer operations.

The *cleansing of the eye* immediately before the operation has been done in many ways. The conjunctival sac, especially the upper fornix, has been syringed with bichloride of mercury and other solutions. Gradually the use of antiseptics has been reduced or given up, and simpler methods have proved just as efficient and less harmful. For twelve years I have used this simple method. The eye and its surroundings are washed with soap, then with a solution of bichloride of mercury 1 to 5000. Particular care is bestowed on the ciliary edges of the lids. Then the lids are everted and the conjunctiva is washed with a piece of absorbent cotton dipped in the same bichloride solution, and the eye is ready to be operated on.

Of the many kinds of dressings after the operation we may get along with two:

(1) The *monoculus*, and for the first days the *binoculus*, the old figure-of-eight bandage, is the best when we have to deal with reasonably quiet

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patients and want immobilization of the eye as much as is possible, or a certain degree of pressure.

(2) The closed eyelids are covered with a piece of wet corrosive sublimate gauze; upon this a wet pad of absorbent cotton is placed so that it holds the lids together like a splint. This cover is fastened by two vertical strips of isinglass plaster, and a third, which covers the upper ends of the strips. When this dressing has dried, in about one-half hour, it is very firm, is simple, and does not irritate. Strips of isinglass plaster placed directly on the lids, according to Professor Arlt, who tried a number of bandages on his own eyes, are the most uncomfortable of all. Patients on whom I employed them complained of their stiffness.

§ II. OPERATIONS ON THE IRIS.

The principal operation on the iris is **Iridectomy,** the excision of a piece of that membrane.

It is indicated

(1) In *closure or obstruction of the pupil* to make a new opening for the rays of light (an *artificial pupil*). This indication is absolute if the eye is otherwise in good condition.

(2) In *central opacities of the cornea and lens.* This indication is limited, for many such opacities let a sufficient amount of regularly refracted rays through, so that sight which was central before the iridectomy is no better, and not rarely worse, after it. To avoid such a disappointment we must carefully ascertain before the operation what sight we may expect from it. This is done by dilating the pupil with atropine and letting the patient see through a stenopæic slit held in different directions before the dilated pupil. If sight is thus materially improved, an artificial pupil is indicated, and it should be done in the place where the vision is best.

(3) In high degrees of *keratoconus* iridectomy has been done to make a lateral, less abnormally curved portion of the cornea available for sight. The artificial pupil will be of greater benefit if the point of the conus has been removed by galvano-cautery, the knife, or a caustic that leaves a dense scar. If the scar is as large as an ordinary pupil, iridectomy is indicated.

Iridectomy made for improving the vision as in the above categories is called an *optical iridectomy.* Iridectomy is, however, very useful as a therapeutic measure, the *curative iridectomy,* and is indicated in the following affections.

(4) To *reduce increased eyeball tension.* The late Albrecht von Graefe, of Berlin, discovered this beneficial action of iridectomy in 1855. After having found that partial staphylomas of the cornea flatten when iridectomy is made, he imagined that iridectomy, reducing the intra-ocular pressure, must be beneficial in glaucoma. His supposition proved true. According to my experience, *partial staphylomas* flatten only in a certain percentage of the cases, but the cure of *glaucoma,* especially the acute forms, by iridectomy has made Graefe a benefactor of mankind.

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(5) To cure or improve *chronic iritis* and *irido-cyclitis* and its sequels. In this group of cases iridectomy, apart from its optical benefit, is a useful means to cut short, in many cases, the frequent relapses of inflammation, and frequently save the eye from impending ruin, as, *e.g.*, in the so-called crater-shaped pupil. In this variety the indication for an iridectomy is absolute, for by re-establishing the communication between the anterior and posterior chambers it saves and frequently improves what sight is left, and if none is left it stops the relapses of inflammation.

(6) In the presence of *tumors or foreign bodies in the iris.* Foreign bodies embedded in the iris may, however, in many cases be removed by letting the implicated portion of the iris fall out through a corneal section, picking up or scraping off the foreign body and reducing the iris.

(7) In *ripening immature cataract* by trituration, according to Förster and others.

(8) As a *preliminary* step to operations for cataract.

(9) In removing *traumatic or operative prolapse of the iris,* especially after simple extraction of cataract. In traumatic prolapse, I do not consider abscission of the protruding iris as absolutely indicated. In perforating wounds of the cornea and adjacent sclera without injury to the crystalline body, I have observed that the protruding iris gradually sloughs off, leaving a clean scar. Prolapses through a corneal ulcer, especially in infective disease, should not be cut, for the iris blocks the passage-way for the entrance of bacteria into the interior of the eye. A fresh traumatic prolapse of the iris should, however, be cut, if we can make an iridectomy free from entanglement of iris in the corners of the wound. After twenty-four hours this is sometimes no longer possible, however carefully we endeavor to reduce the columns of the coloboma.

The instruments required are:

(1) A *lid speculum.* (Fig. 2.) The wire specula with a screw are in general use and answer the purpose. One will do for both eyes.

(2) A pair of *fixing forceps.* (Fig. 3.) They ought to be accurately made, closing readily, and opening without a jerk. They should be applied quite near the corneal margin, the teeth penetrating into the episcleral tissue. Held in this way they steady the eyeball well enough, making the ophthalmostats with a double pair of prongs unnecessary. They ought to steady the eye without pushing or pulling, moving it in the direction of the tangent of the point of insertion. The pair illustrated in Fig. 3 can be taken asunder and perfectly cleansed and sterilized.

(3) A *bent lance-shaped knife.* (Fig. 4.) I have all my lances bent at the same angle, to which I have accustomed myself. If the bend differs we have to think and adjust ourselves to the particular angle. Any motion which we have to make frequently soon becomes instinctive, and we make it correctly without thinking. In the short time occupied by an eye operation we have to observe and consider many things which, though small, are important. A straight lance for an iridectomy on the temporal side

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may be added to our armamentarium, as it is convenient also for other purposes.

(4) A pair of *curved iris-forceps.* (Fig. 5.) They are apt to be faulty in two points: (1) The tips of the branches have sharp edges instead of being rounded off. This makes them scratch the surface of the cornea or engage in the iris. (2) The tips of the branches close on moderate but diverge on stronger pressure, letting slip the iris, which they had grasped before.

(5) A *blunt* (Tyrrell's) *iris-hook* (Fig. 6) for cases where the lens is absent or dislocated.

(6) A pair of *curved* or *straight iris-scissors* (Fig. 7), delicate but well-closing and sharp.

(7) A combined *spatula* and *blunt-pointed probe.* (Fig. 8.)

(8) A *narrow-bladed cataract-knife* (Graefe's). (Fig. 9.)

(9) Mathieu's *iris-forceps.* (Fig. 10.)

(10) Narrow-bladed but strong strabismus-scissors (Stevens's). (Fig. 11.)

EXECUTION OF THE OPERATION.

We describe an upward iridectomy. The patient lies reclining on the operating chair; his eye is sterilized and cocainized. The ambidextrous operator stands behind and a little to the right side of the patient, whether the right or the left eye is to be operated on. He holds the lance-shaped knife in his left hand, between the thumb on the front side and the index- and ring-fingers on the other. His right steadies the eye with the fixing forceps inserted into the conjunctival and episcleral tissues with a broad, deep grasp. The patient looks slightly downward. The operator puts the little finger of his left hand on the forehead of the patient, applies the point of the lance to the limbus (transparent margin) of the cornea, thrusts the blade first slightly towards the iris, but as soon as he recognizes by the lustre of the blade that he has entered the anterior chamber, he turns the handle backward and pushes the blade towards the axis of the eye parallel to the surface of the iris. When he has entered the knife far enough to obtain a corneal incision as large as he desires, he withdraws the knife, taking care neither to grate on the cornea nor to wound the lens. He now intrusts the fixing forceps to an assistant, takes the iris-scissors in his right hand and the iris-forceps in the left. He enters the branches close to the pupillary edge of the iris, separates them, seizes the iris in joining them, draws it straight out, and cuts the protruding part with one stroke of the scissors. If the iris has not lost its elasticity, the sphincter corners will return to their natural position, and the pupil will have the appearance of a key-hole. Should this not be so, parts of the iris are incarcerated in the angles of the wound, and have to be reduced either by rubbing over the edges of the wound and adjacent cornea with a spatula, or, if this does not liberate the iris, by entering the blunt probe into the anterior chamber to disengage the entangled iris. It is important that an artificial pupil

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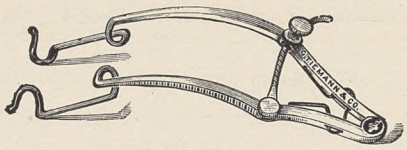
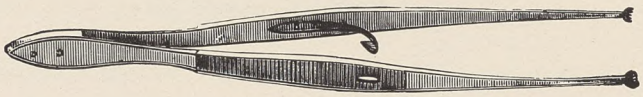
FIG. 2.



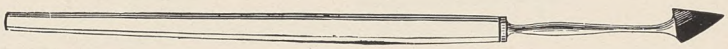
Lid speculum.

FIG. 3.



Fixing forceps.

FIG. 4.

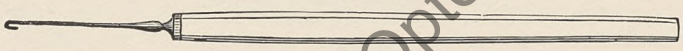


Bent lance-shaped knife.

FIG. 5.

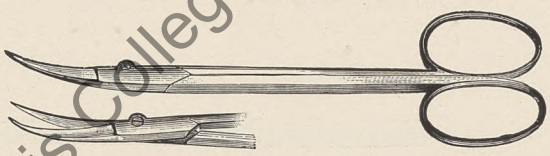
Curved iris-forceps.

FIG. 6.



Blunt (Tyrrell's) iris-hook.

FIG. 7.



Iris-scissors.

FIG. 8.

Combined spatula and blunt-pointed probe (flexible).

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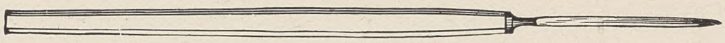
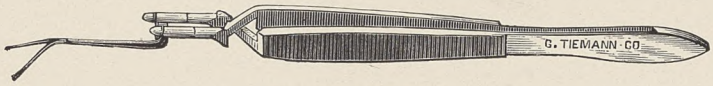
FIG. 9.



Narrow-bladed cataract-knife (v. Graefe's).

FIG. 10.

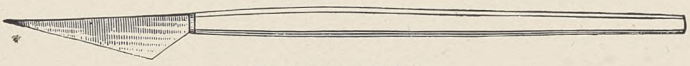


A Mathieu iris-forceps.

FIG. 11.

Narrow-bladed but strong strabismus-scissors (Stevens's).

FIG. 12.



Beer's cataract-knife.

FIG. 13.

Cystotome, straight or bent.

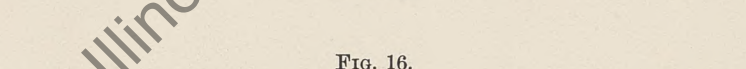
FIG. 14.

Daviel's spoon, usually combined with cystotome.

FIG. 15.

Wire loop.

FIG. 16.



Knife-needle.

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should be clean,—*i.e.*, the wound free from the least incarceration of the iris.

During the operation a few drops of a mild antiseptic lotion are let fall on the wound and the cornea, more with a view to keep the cornea bright than to keep the wound aseptic.

The eye is dressed and the patient put to bed.

In an ordinary iridectomy, such as we are supposed to make, for instance, preliminary to a cataract operation, there is very rarely any reaction, and the patient can be discharged in a week or sooner.

MODIFICATIONS OF THE TECHNIC OF THE OPERATION.

The operator is right-handed, but not ambidextrous. He may use the lance-shaped knife with his right hand, steady the eye with the left, and

(1) proceed exactly in the same way as described above, only standing to the left of the patient's head.

The lance may be held in two ways different from the above-described, both being perfectly good.

(2) The *lance is held like a writing-pen.* This modification may be the most natural, for we all know how to hold a pen, and I see it in many drawings.

(3) The operator stands behind the patient's head, and the lance is held as above, between thumb and index- and ring-fingers, but *the latter are in front.* This modification seems awkward to me, but I have seen very good operators use it.

In iridectomies it matters little how we hold the instrument, but in cataract operations the one first described, the old classical manner, is the best. I do not know how I should execute correctly a discission of a secondary cataract in any other way. This is the reason why I hold all cutting instruments that have to be introduced into the eye in the same way.

Instead of the lance a *narrow-bladed knife* (Fig. 9) may be used for making the incision. This is a decided advantage in glaucoma and wherever the anterior chamber is shallow. For accuracy in determining the length of the section, which in optical is less than in curative iridectomies, the lance is preferable.

MODIFICATIONS OF THE IRIDECTOMY BY THE REQUIREMENTS OF THE DISEASE.

(1) The *glaucoma iridectomy should be large and peripheric.* A Graefe knife is thrust through the scleral border of the anterior chamber one millimetre or a little more behind the limbus. The section should be no smaller than one-fifth of the corneal circumference. The iris should be cleanly cut by two or three successive strokes of the scissors, so that one stroke cuts the iris at the temporal corner, the other the middle portion, the third that at the nasal corner of the section. This refers to the right eye; for the left the order is reversed.

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(2) When the *pupil is closed by a pseudo-membrane, or a circular synechia ties the pupillary edge of the iris to the lens*, the iris-forceps should be pushed forward to the very edge of the pupil, then opened without receding; in closing the sphincter portion should be seized and drawn out. In case this is not done, the pupillary portion will remain in the eye. If so, the chief object of the operation is not frustrated, as the communication between anterior and posterior chambers is re-established through the peripheric coloboma; but if cataract be present or should develop later, the remaining portion of the iris would somewhat complicate the operation.

(3) If we want to make a *very small pupil* for *optical* or *other purposes*, *e.g.*, a so-called *sphincterotomy* when the pupil appears insufficient for the exit of a cataract, we make a small corneal incision about two millimetres from the limbus, draw the iris out with Mathieu's (Fig. 10) forceps, and cut off as little of the iris as we think proper by closing the iris-scissors at right angles to the corneal incision.

The Mathieu *forceps* are in their mechanism an improvement on a pattern of Liebreich. The short branches have their teeth at the lower surface instead of at the end; they are in contact when we compress the handles, diverge when we reduce the pressure, but invariably remain in contact at their crossing-point. They can therefore be introduced through a very small opening and yet grasp a considerable piece of iris. They require good workmanship, but are very valuable, and last a long time.

(4) *For glaucoma in an aphakial eye*, as it develops in about one per cent. of the cases after discission of secondary cataract, and no less after extraction of primary cataract, we make an incision with a lance-shaped knife in the opaque border of the cornea, but we nearly always fail to grasp a piece of iris with the ordinary forceps; we may succeed with Mathieu's forceps, but the surer instrument is a blunt hook (Fig. 6, facing page 784). The anterior chamber in most of these cases is filled with vitreous, of which a bead commonly protrudes after the incision. The hook (or forceps) is introduced through it into the pupillary area, its curved tip behind the edge of the iris, and drawn out with the piece held in its grasp. The iris is cut close to the wound, and the eye bandaged. The recovery has been short and good in all the cases I have operated on,—about twenty.

ACCIDENTS AND MISTAKES DURING THE PERFORMANCE OF IRIDECTOMY.

(1) When the anterior chamber is very shallow we may, for fear of wounding iris and lens, *advance the blade only between the lamellæ of the cornea*. If we use a lance, this may be recognized by the lack of the metallic lustre which distinguishes that part of the blade which is in the anterior chamber. If we use a Graefe knife, we can also directly notice its appearance in and passage through the chamber. In both cases, if we fail to enter the anterior chamber no aqueous escapes, and the iris-forceps are stopped in the corneal wound-canal. Should delay of the operation be inadvisable, we have to introduce the instruments again, correcting our mistake. The recovery will not be materially prejudiced by the double cut.

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(2) When we have entered the anterior chamber the knife may *engage in the tissue of the iris.* In most cases slight lateral and forward movements with the knife will make the point free again. If we do not succeed in our attempt, the safest thing is to withdraw the knife, but at the same time enlarge the corneal wound by cutting from within outward. Should after that the wound be still too small, it can easily be enlarged with a fine but strong pair of curved scissors. (George T. Stevens's strabismus-scissors (Fig. 11) are excellent for this purpose.)

(3) *Injury of the capsule of the lens.* This accident, which is followed by cataract,—unless the opening be very small,—occurs when during the advance of the lance-shaped knife or the iris-forceps the patient by a sudden, unexpected movement runs the point of the instrument into his lens. If the lens was not cataractous before, the damage can be repaired only by subsequent removal of the lens. Let us always bear in mind the possibility of this accident, and be on our guard; but let us blame nobody for it, and keep our lips sealed when a patient is presented to us who had an iridectomy made for glaucoma, whereupon a cataract developed, which by the rent in the capsule we recognize as being traumatic. The by-standers hold their eyes and ears wide open, and, on mischief bent, construe each word and look in condemnation of the operator.

(4) *Rupture of the capsule* may also be produced, not by an untoward movement on the part of the patient, but *during the attempt to seize the iris with the forceps.* This is particularly to be feared when in cases of old hemorrhagic glaucoma the iris is atrophic and blood fills the anterior chamber.

(5) *Hemorrhage into the anterior chamber* may have two causes: a faulty technic in the operation and a hemorrhagic predisposition of the eye operated on. As to the first cause, the fault commonly lies in an iridodialysis, either during the onward movement of the iris-forceps in the anterior chamber, when a clumsy or ill-finished instrument (see page 784) dragged the iris onward, or when, during the drawing outward of the iris, the traction was too strong or oblique, so as to tear the iris away from its insertion. I noticed this in my earlier practice, and have carefully avoided it since. It may also be caused by an imperfect pair of iris-scissors, especially when it is pushed during the cutting. As to the second cause, the hemorrhagic diathesis of the eye, it is but too frequently encountered in practice. We see immediately after the corneal incision, before the iris is touched, blood issue from the iris in points scattered over its surface. In many cases this will not lead to profuse bleeding; it soon stops, and the operation is continued and finished without further hemorrhage. In other cases, however, especially in glaucomatous eyes, there is free bleeding when the iris is cut. When the whole anterior chamber is filled, I commonly press some blood out with a spatula until I see the periphery of the iris; then I bandage the eye.

(6) *Prolapse of vitreous* may occur during the stroking of the iris out of the corners of the wound; also by the sudden diminution of the intra-

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ocular pressure on the escape of the aqueous. It has usually no bad consequences, so that certain operators, for instance, the late Dr. W. Roeder, of Strassburg, recommended it as a favorable step in the glaucoma operation.

(7) *Incarceration of iris in the corners of the wound.* This is one of the commonest imperfections of the operation, which even with the greatest skill and care can only be reduced in number. In healthy irides it can mostly be avoided; in acute, especially consecutive, glaucoma, we must at times be contented with a small, pear-shaped coloboma, the columns of which adhere to the section. We have to do this first operation in order to cut short the deleterious action of the disease on the eye, and can make another later, should the first not insure a permanent recovery.

PROCESS OF HEALING.

The common course of recovery is permanent union of the wound from the first night, some congestion in the neighborhood of the wound for a few days or longer without any significance, cornea, iris, and media clear, the pupil responsive as before. The cicatricial contraction of the scar produces a low degree of astigmatism, augmenting the refraction of the meridian parallel to the incision.

The process of healing is disturbed by the original disease in many ways.

In cases of plastic iritis we commonly obtain good and permanent recovery.

In crater-shaped pupils the same.

In irido-cyclitis, especially in that caused by sympathy, the pupil commonly closes again by the continuance of the original inflammatory process.

In glaucoma we *mostly* have undisturbed recovery where there is no degeneration in the inner tunics of the eye, though a *slow and imperfect restoration of the anterior chamber* is not uncommon. When the absence of the anterior chamber is not connected with pain and inflammation in the eye we need have no particular misgivings, but should keep the eyes bandaged and the patient in bed as long as he gives us no cause for apprehension on the part of his lungs or vascular system. A permanently imperfect restoration of the anterior chamber is mostly accompanied by a gradual failure of sight.

Malignant glaucoma is called a wound reaction which almost[1] always terminates in blindness. An ordinary iridectomy in a case of chronic glaucoma is followed in from two to twelve hours by intense pain, lacrymation, œdema of conjunctiva and lids, abolition of anterior chamber, cornea hazy, pupil and iris dull, fundus invisible, T +2 or +3, and rapid extinction of the sight that was left; in short, the operation has converted a case of chronic glaucoma into one of fulminant glaucoma. Sclerotomy, pressing back of the lens, removal of the lens, and internal remedies have been of no

[1] See the elaborate paper on this subject, with the report of a favorable case, by Dr. H. Friedenwald, in Archives of Ophthalmology, 1896, p. 478.

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avail.[1] In the course of weeks or months the wound becomes ectatic, fistulæ form, which, upon breaking, give the patient relief from the pain, to suffer again when, on closure, new attacks set in, until the eye bursts and shrinks, or is enucleated. It is an awful disease. Schweigger, who published two excellent papers on it,[2] says these eyes are all lost, and, moreover, as the one eye has gone, the other is sure to go when operated on. I can confirm this statement by about half a dozen cases from my own practice. The cases are rare, and in some that recovered the picture was not complete, and I had to revise my diagnosis. Yet there is a border-line of intensity in the same disease beyond which recovery is not known.

Another disturbance of the healing process are the *hemorrhages repeating themselves indefinitely* in diabetics and other persons of a weak constitution. It is remarkable that some patients of this group preserve available sight for many years after the operation, in spite of incessant relapses of retinal hemorrhage.

Loss of an eye by suppuration from an iridectomy, which thirty years ago, according to Arlt, occurred once in two hundred and fifty cases, is now almost unheard of.

RESULTS OF IRIDECTOMY.

The benefits derived from iridectomy were greatly overestimated thirty and forty years ago, the time when by his brilliant talent A. von Graefe easily conquered for it the recognition of the medical profession everywhere. Its easy execution, its comparative freedom from danger, and its saving of sight in many cases which formerly were utterly hopeless, brought it at once into general favor among ophthalmic surgeons. It was a grand revival of a good old operation. But the reaction has not been missing. The disappointment in the expected gain of vision from iridectomy in corneal opacities soon reduced the large number of operations on this line to a very small percentage. Even in glaucoma we look on our operative successes with much less pride than formerly. And yet it still is **one of the most beneficial operations on the eye.** Nothing can equal in effect the "wonderful" restoration of sight when so simple an operation as iridectomy opens the path for the rays of light through a congenitally closed pupil. I beg leave to illustrate this by a case of my earliest practice, which I have never published, but which created a great sensation among the general public.

A poor widow brought to me her twelve-year-old daughter, who, she said, never had any inflammation or disease in her eyes, but was born blind. I found both pupils narrow and closed by a pseudo-membrane. One eye showed other signs of irido-choroiditis, was shrunken, and totally blind. The other, however, was well formed and had good perception of light. My diagnosis was congenital blindness from fœtal irido-choroiditis. I admitted her to the clinic, and on one eye made an iridectomy which gave her

[1] Friedenwald treated his case with large doses of salicylate of sodium, gr. xx every three hours for twelve hours. Great depression, but marked improvement in eye.

[2] Archives of Ophthalmology, vol. xx. p. 475, and vol. xxv. p. 222.

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good sight. The case was presented and analyzed before the students as remarkable and important in its embryological and practical aspects. She left the hospital in a week.

Two weeks later the secular press was full of high-colored accounts of the "wonderful restoration of sight in a blind-born twelve-year-old girl by the *Holy Virgin of Walldürn*," the Lourdes of the Catholic portion of the Grand Duchy of Baden. The wonder was attested by the priest, the physician, and the mayor of the place, and by a Spanish count. The incident was true, the story went, and the wonder so much the greater as the child had been admitted to the Eye Clinic at the University of Heidelberg and after short treatment discharged as incurable. As soon as I read the story I informed the priest of the facts. The report had been so widely circulated that a high official of the government sent me a letter of inquiry in regard to what I knew about the case. I answered that I had written to the priest at once, but had not received a reply. I had abstained from a public refutation because I believed the priest to be a benevolent, honest man, for he had sent to the University clinic many eye patients that had gone to Walldürn. The government official wrote me back, "You will receive your justification, but you will have to wait two weeks. The place requires a wonder every few years to keep up its reputation." So it was. The priest thanked me that I had undeceived them, and in excuse for the delay of his answer he said that the child had been presented to them as congenitally blind, and had behaved so, but after she had been led to the church several times, and then held over the altar of the Holy Virgin, she had exclaimed, "I see! I see!" In order to explain the undeniable fact of the child's sight they had written to the mayor of the birthplace of the child, and had not received an early reply.

The *results of iridectomy in glaucoma* are both of the most brilliant and of the most gloomy kind, according to the nature of the case. The almost invariable permanent restoration of sight in the acute cases with preservation of the accommodation is paralleled by few operations. These cases form the great minority in the whole group. The subacute cases give fair results, mostly the sight present at the time of the operation is preserved, and sometimes the progress of the glaucoma is arrested only after a *second iridectomy*. Graefe recommended to make the second iridectomy opposite the first. I have mostly made it at one side of the first, so that both appear as one larger operation. The results have been as good as, perhaps better than, when the two were opposite. In case a second operation has not been satisfactory, a *sclerotomy* may be done (see later on).

In spite of these operations, the majority of patients with *chronic glaucoma*, and almost all with *simple glaucoma*, will gradually lose their sight; the myotics may retard the failure, but will not ward it off. In general we may express the results of iridectomy as good in acute, fair in subacute, palliative in chronic, poor in simple glaucoma.

Hirschberg, Nettleship, C. S. Bull, and others have published statistics on the results of iridectomy in glaucoma. They are serviceable, but not so valuable as those of cataract and other operations, as it is too difficult to follow up a sufficient number of successive cases. Glaucoma is not an idiopathic disease, but a variegated picture resulting from the presence of one more or less pronounced symptom occurring in different morbid conditions. The iridectomy will ameliorate the symptom, but it may fail to have any influence on the primary disease. Its results and the prognosis in each case must, therefore, be based on the primary disease, be it general or ocular, and on the changes produced in the eye by the degree and dura-

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tion of the hypertony, a complicated problem which we must try to solve step by step. Take as an example hemorrhagic glaucoma, which is generally dreaded. Schweigger, whose authority on glaucoma nobody will question, says, "Make iridectomy in this as in every other kind of glaucoma; hemorrhagic glaucoma is not so bad as its reputation." That may be so in general, but the prognosis of the case will vary with the etiology of the hemorrhages and the structural changes present in the eye under observation. Ocular hemorrhages, with no cupping or atrophy of the optic disk, fair sight, complete field but a disorganized iris, and slight increase of tension, constitute, in my opinion, about as unfavorable a case as we can have to deal with. If we make an iridectomy, there will probably be more hemorrhage and an acute outbreak of glaucoma. If we wait until the acute attack sets in spontaneously, an iridectomy will produce a poor coloboma and flood the anterior chamber and probably also the vitreous with dark blood. The patient will be worse instead of better, and the vision may be destroyed. The blood absorbs, the optic nerve is neither atrophic nor cupped, retinal hemorrhages appear now and then, the tension remains increased in spite of myotics, and the sight is permanently lost. The other eye shows the beginning of the same affection: good sight and field, no abnormity in fundus, T + 1 periodically, no subjective symptoms, but the *ominous rotten iris*, with a wide, oval, immovable pupil. I shall not advise this patient to be operated on, but treat him with pilocarpine. This picture is only slightly composite: all the features except the iridectomy before the acute outbreak were present in one case.

OTHER OPERATIONS ON THE IRIS.

Among the operations on the iris which have a more restricted application we mention the following:

(1) **Iridotomy** or **iritomy**, an old operation revived and perfected by L. de Wecker, of Paris. It is used when after removal of the lens the pupil is closed. Its object, to make a new pupil by cutting through the iris, formerly was obtained by making with a narrow knife or a broad needle an incision into the iris, the tissues adherent to its posterior surface, and the vitreous. The direction of the incision had to cross the iris fibres, so that the contraction of both lips towards the periphery will keep the perforation open. The results frequently were unsatisfactory, and the operation is little used now.

De Wecker performs the operation in the following way. He makes an incision into the cornea with a lance, say near the lower border, if the iris is drawn up, then he introduces his pinces-ciseaux into the anterior chamber, transfixes the iris with the pointed branch, and advances both branches, the one behind, the other in front of the iris, obliquely up and nasally. After cutting the iris lying between the branches in this direction, he draws the instrument slightly backward and makes another incision through the iris diverging from the point of entrance upward and tem-

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porally, thus cutting out of the iris an angular tongue, which will roll up towards its base.

De Wecker in certain cases makes with the same instrument only one incision through the iris, crosswise to the fibres. I have seen de Wecker and other surgeons make iridotomy in each of these ways with satisfactory results. I have tried it in several cases, but, getting more reaction and less benefit than I expected, I have not cultivated the method.

(2) **Irido-cystectomy** is the operation which, from the beginning of my practice up to to-day, I have used as the chief method for making a new pupil when irido-cyclitis or irido-capsulitis, after cataract operations or traumatisms, had closed the old. The cases in which it is indicated are not very numerous, but they occur in a very small percentage of cataract operations, and I have treated them with more satisfaction by this method than by iridotomy. *It is performed as follows.* Under cocaine anæsthesia a Beer's cataract-knife (Fig. 12) pierces the cornea about three or four millimetres above the lower corneal margin, opposite the scar from the extraction, and transfixes the iris or pupillary pseudo-membrane by an opening three or four millimetres long. The knife is withdrawn. With a blunt hook (Fig. 6) the lower lip of the iris wound is seized, drawn out of the eye, and abscised close to the cornea. Eye bandaged. There are scarcely any accidents worth mentioning. The healing is usually prompt, and yields mostly surprisingly good visual results. Among the failures after this operation I have noticed reclosure of the new pupil and suppuration, but they are rare. The infection in the cases of suppuration must be ascribed to the waking up of pyogenic microbes which had entered the eye at the first operation and lain dormant in the inflammatory products of the iris and capsule, for an infection by the instruments can easily be excluded. Infective material may, however, be carried into the eye from the conjunctiva by prolapse of the vitreous, which is not infrequent during this operation.

(3) **Corelysis, Synechiotomy.**—An operation revived and particularly cultivated by the late Mr. Streatfeild, of London, in 1857 and later. For a time it was quite popular, owing to the current belief that the posterior synechiæ exerted a constant and pernicious irritation on the eye, on account of their pulling at the iris and its nerves and vessels during the play of the pupil.

Technic.—Through a small corneal incision a blunt probe was passed into the pupil behind the synechiæ, which, one by one, were gently detached from the capsule. There were scarcely any accidents to be recorded of this operation. The results were not conspicuous. In a number of cases some of the synechiæ could not be loosened, in others they reattached. The therapeutic and visual gain was little. The operation, which when a student in London I witnessed many times, has fallen into disuse.

(4) **Iridodesis, iridesis,** tying a piece of the iris outside a small corneal wound. This was a modification of the old operation of **iridencleisis,** by George Critchett, of London, in 1857. Both had the object of drawing

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the pupil away from opacities in the cornea and lens situated in the region of the axis of the eye. This occurs sometimes spontaneously after a peripheric perforating corneal ulcer. The optical effect seems to be good, but not enough to outweigh the risk from the operation. *Iridencleisis* is safer than iridesis. A small perforation was made near the periphery of the iris, and a small piece of iris drawn into it and left there. The portion locked up in the puncture canal became agglutinated to the cornea, forming an anterior synechia; the protruding part sloughed off.

Iridodesis is done in the same way, with the addition that the artificial prolapse was tied with a delicate silk thread, for which purpose Critchett had devised a special instrument. I saw him do a great many of these operations in 1857. When afterwards I went to Graefe, in Berlin, he asked me, "What new things have you seen in London?" I told him of Critchett's iridesis. He shook his head and said, "I shall not do any of these operative subtleties. A clean, simple iridectomy is good enough for me." The operation had a certain run; I made several myself, but they were brought to an untimely end by a case that developed irido-cyclitis glaucomatosa, for which I had to make an iridectomy. The operation is out of use.

§ III. OPERATIONS ON THE CRYSTALLINE BODY.

Operations on the crystalline body are indicated in all cases in which opacities of it, *cataracts*, intercepting the rays of light, can be removed to the benefit of the patient. They are not indicated when the opacities are so small (*partial cataracts*) as not to interfere with fairly good sight. Cataract operations are of three kinds: *displacement*, where the cataract remains within the eye but is pushed away from the pupil; *extraction*, where it is taken out of the eye; and *discission*, where the lens capsule is divided, and the lens substance brought in contact with the aqueous humor, by which it is gradually dissolved and absorbed. This kind of operation is therefore also called the method by *solution*.

All these methods primarily refer to the lens proper. When the latter has been put out of the way there commonly remain portions of it and the capsule behind, more or less obstructing the pupil. These remnants, to which products of inflammation may be united, are called *secondary cataracts*, which also must be dealt with. We have, therefore, to operate for *primary* and *secondary cataracts*, for which, according to special indications, one of the above methods is selected.

A. OPERATIONS FOR PRIMARY CATARACT.

I. DISPLACEMENT.

Displacement, the oldest method, has now more historical than practical importance. It is described by Celsus and other authors of antiquity, and held the only place until 1745, thenceforth a forward place until about 1850, when it began to lose ground, and rapidly disappeared from the stage. It was done in two ways.

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(*a*) By *depression* (abaissement, keratonyxis). A broad needle is introduced through the lower segment of the cornea into the pupillary space, with the surface of the blade flat upon the capsule of the lens, then by gradually raising the handle of the needle the lens is pressed down and couched in the lower-anterior part of the vitreous. The needle is withdrawn and the patient put to bed.

(*b*) By *reclination* (couching, scleronyxis). The needle is introduced through the sclerotic, about seven millimetres behind the cornea and five millimetres below the horizontal meridian on the temporal side. It is thrust obliquely forward and upward, passing through the lens into the upper part of the pupil. Then with the flat of the needle the upper part of the lens is "reclined," and the whole pressed down into the anterior-lower part of the vitreous.

Accidents and Results.—The cataract would often rise again before the needle was withdrawn, and the couching had to be repeated several times before the cataract kept its new position. It mostly rose again the next day or some time later. It produced irido-choroiditis, glaucoma, detachment of the retina, and phthisis bulbi.

The immediate, and in some cases—we do not know the percentage—the permanent, result has been excellent. The lens remained in its new bed, the cortex was absorbed, the nucleus adhered to the corona ciliaris without causing trouble. The bad cases outnumbered the good ones, and the general results were below those obtained by extraction, so it was gradually given up entirely. In 1858 I heard A. von Graefe say that he considered reclination indicated when in old, decrepit, or otherwise ill-constituted people one eye had been destroyed by suppuration, making it likely that the other would follow the same course. I did two reclinations in my own practice, being disgusted with the horrible mutilation and the unpleasant involvement of the ciliary body by the Jacobson method of extraction (large peripheric section with broad iridectomy) then (1861) in vogue.

Reclination has still its followers in certain cases of shrunken and secondary cataracts, which the pupils of the Vienna school—Fuchs, Elschnig, and others—advise to dislocate either by keratonyxis or scleronyxis. Even in such cases, I think, it will be surer and safer to extract or divide them through the cornea. I have, at least, not come across a case of secondary cataract where I would have given preference to displacement over discission or extraction.

II. EXTRACTION.

Historical Introduction.—This operation, the greatest in ophthalmic surgery, and one of the most memorable in general surgery, was invented by the French surgeon **Jacques Daviel**, in 1745.

How he was led to it he relates in a paper read before the Académie Royale de Chirurgie, November 16, 1752, entitled "Nouvelle Méthode de guérir la Cataracte par l'Extraction du Crystallin," and published in the "Mémoires de l'Académie de Chirurgie," édition de 1769, t. ii. pp. 337 to

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354, with two very fine plates. I shall give it in extract taken from a very interesting paper by Dr. D. E. Sulzer, in the *Annales d'Oculistique*, November and December, 1895, entitled *Documents servant à l'histoire de la Cataracte. Essai historique.*

A hermit, who had lost one eye by depression of cataract, came to me at Marseilles, in 1745, for the operation on the other eye. I made a keratonyxis with an ordinary needle sharp on both sides. I did not succeed in couching the cataract, pieces of which fell into the anterior chamber. The latter filled with blood, and I had to withdraw the needle. This accident determined me to follow the example of Mr. Petit.[1] I opened the anterior chamber with a curved needle and enlarged the wound with curved scissors. All the contents of the anterior chamber, blood and cataract, escaped, the pupil became clear, and the patient recognized objects held before him. The eye was lost by suppuration.

This case, though unsuccessful, determined me always to open the anterior chamber, seek the lens in its case (*chaton*), make it pass through the pupil into the anterior chamber, and draw it out of the eye.

I made this operation for the first time on a woman. Opening the anterior chamber as in the above case, I introduced a small curette into the pupil, detached the cataract, and removed it in pieces out of the eye with the same instrument. The pupil appeared clear, the patient experienced not the least accident, and was cured in two weeks.

Daviel's operation, which he modified on further experience, was at once taken up by surgeons of every country and tried in all directions, as it has been up to the present day. Of these modifications and the results obtained by Daviel and others, more later on.

INDICATIONS.

Extraction is indicated (*a*) *unconditionally in all hard cataracts when the functional examination warrants the possible restoration of good vision.* There must be *perception of light* from all parts of the field of vision. A fully *ripe* cataract, *i.e.*, a lens all the portions of which have become opaque, transmits enough regularly refracted rays to locate, by alternate covering and uncovering in a dark room, a lighted candle at a distance of from eight to twelve feet directly in front, and from two to four feet in all other places of the field of vision. We say, "*light perception and projection normal.*" There are many cataracts for which extraction is indicated, absolutely or conditionally, though they do not answer the above conditions of maturity, light perception, and projection. As to *maturity*, it is certainly desirable to have to deal with a ripe cataract, but often enough there are patients whose cataracts mature so slowly that we have to remove them of necessity. We have then before us the alternative of ripening the cataract artificially or removing an immature cataract.

For artificial ripening a number of operations have been recommended and practised.

(1) *Discission*, the most efficient of them, falls short by ripening only the anterior cortex, so that considerable portions are apt to be left behind,

[1] The celebrated French surgeon Jean Louis Petit had in 1708 made a corneal section to extract a cataract which had fallen into the anterior chamber. (Mémoires de l'Académie Royale des Sciences, 1708.)

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although the cataract appeared mature, and the extraction was done carefully and, as it seemed, totally. Disappointments in this respect have repeatedly surprised me, even when the pupil appeared black and the patient immediately after the operation counted my fingers readily.

(2) *Massage of the lens*, according to Förster. An iridectomy is made, and the lens triturated by rubbing a blunt probe over the cornea.

(3) Paracentesis of the cornea is made, and the lens *triturated through the cornea without making an iridectomy.*

(4) Paracentesis of the cornea or iridectomy is made, and the lens *directly triturated with a blunt instrument* introduced into the anterior chamber through the opening in the cornea.

All these procedures have the disadvantage of being, in a number of cases, either totally or partially inefficient, besides adding to the removal of the cataract another surgical procedure which has not always proved harmless.

Many operators—the present writer included—prefer the risks of removing an immature cataract to any ripening operation.

Maturity and operability are not always interchangeable terms.

A *cataract may be fully mature and yet not in the best stage to be removed;* for instance, the cataract with asbestos-like sectors and that swollen by imbibition are unfavorable to operate on. Their cortices adhere fast to the capsule, requiring inordinate manipulation to get them out, and it is difficult to pass the knife through the shallow anterior chamber without injuring the iris, difficult also to make the counter-puncture and the section geometrically correct.

On the other hand, *a cataract may be immature and yet perfectly fit to be removed.* Schweigger and others have made the observation that immature cataracts in elderly persons, fifty-five years and over, can be expelled totally. This, according to the experience of the author, is true in the majority of cases; in a certain number, however, considerable cortex remains in the eye, which, though it can be dealt with by subsequent discission, is always a disappointment to the patient and unpleasant to the operator.

(*b*) Extraction is indicated *conditionally in complicated cataracts where the possible restoration of good vision is doubtful;* for instance, in partial detachment of the retina, atrophic choroiditis, albuminuric and diabetic retinitis, glaucoma, opacities of the cornea, traumatisms, etc. All these conditions give a bad prognosis, yet they frequently yield results highly gratifying to the patient; for instance, we extract a cataract from an eye suffering from partial detachment of the retina, the other eye being blind. I have seen such operations succeed so that the patient could find his way alone and earn his livelihood by sawing wood or by other coarse occupations for a number of years.

(*c*) Extraction is indicated as *a subsequent operation* to discission in soft cataracts when glaucoma develops; the same in traumatic cataract; further,

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to abridge the recovery when lens masses, cataractous from traumatism or operation, are slow in absorbing.

(*d*) *Shrunken cataracts* and *tough capsules*, occluding the pupil after removal of the lens, can be better dealt with by extraction than by any other procedure.

Considering the **indications in general**, five questions should be discussed:

(1) *Which is the proper age for the performance of extraction?* The age of fifteen years is commonly considered as the end of discission and the beginning of extraction. Old age, even very old age, in itself is no contra-indication, yet greater precaution as to maintenance of the general health is to be taken.

(2) *Shall we operate for cataract as long as the other eye has good sight?* I say, "Yes;" adding that we should operate for cataract when the chances of success are greatest. The risk from a cataract operation as to life's comfort or integrity of the other eye is so small that it does not compare with the gain from restoring sight in one eye. There are only a few people that complain of discomfort from an aphakial eye, and this is far outweighed by the acquisition of a greater field, and in a number of cases by the restoration of stereoscopic vision.

(3) *Is it not only justifiable but desirable to operate at the same time on both eyes, provided the cataracts are both ripe?* I should say, "In general, no." The loss of both eyes is so appalling that an operator whose hands have once been instrumental in such a calamity naturally seeks to avoid it. Furthermore, during the treatment we learn a great many peculiarities of the bodily and mental conditions of our patients which it is of advantage to know at the second operation. Apart from our own feeling, we owe it to the patient not to stake his future happiness on one chance. Let him have the choice of better conditions the next time. On the other hand, there may be particular circumstances making it desirable to have the two eyes operated at the same time. A good compromise is to operate on the second eye a week or two after the successful operation of the first.

(4) *Which season is the best?* This question through the introduction of asepsis and antisepsis has lost its general importance. We may say that fat people should avoid the heat of the summer; that people with lung affections and even those suffering from lacrymal troubles should avoid the cold and damp season.

(5) *Which hour of the day is the best for the performance of an operation* so important as the extraction of cataract? Some surgeons operate early in the morning when they are fresh, and before they have touched any other patient. This is good for the operator, but it is better for the patient to be operated on in the afternoon, because the usual five or six hours of smarting will then be followed by an undisturbed sleep, during which the union of the wound has the best chance to take place and become permanent.

I shall begin with the technic of the so-called simple extraction, which I have practised as a general method for the last eleven years.

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INSTRUMENTS REQUIRED: 1, a wire speculum, Fig. 2; 2, a fixing forceps, Fig. 3; 3, a narrow Graefe knife, Fig. 9; 4, a cystotome, Fig. 13, straight or bent, with an extra fine, delicate cutting tooth, rounded on its base; if bent, one for the right and another for the left eye are required; 5, a Daviel's spoon, Fig. 14; 6, a wire loop, Fig. 15; 7, a spatula and blunt-pointed flexible probe, Fig. 8. If excision of the iris becomes necessary, furthermore: 8, iris-forceps, Fig. 5; 9, iris-scissors, Fig. 7; and if the wound is to be enlarged, 10, Stevens's strabismus-scissors, Fig. 11.

TECHNIC OF EXTRACTION.

First Step: (Upper) Corneal Section.—The ambidextrous operator, standing behind the patient, who reclines on an operating chair, the sterilized eyelids separated by a wire speculum and looking slightly down, inserts the fixing forceps into the conjunctival and episcleral tissue with his left hand, if the right eye is to be operated on, and *vice versa*, and thrusts the point of a narrow knife (Graefe's), the cutting edge directed towards the upper edge of the cornea, through the limbus of the cornea on the temporal side about one-half or one millimetre above the horizontal meridian, pushes it straight across the anterior chamber, keeping clear of the iris, and transfixes the limbus of the cornea on the nasal side at a point just opposite to the point of entrance on the temporal side. Without stopping or turning the knife on its long axis, the operator proceeds to push the blade towards the nose, and at the same time cutting upward always along the limbus on both sides until he is near the upper end of the cornea, where, to avoid a jerk, he slackens the movement of the knife, but without changing its course, completes the section, and forms a small conjunctival flap. The section thus comprises almost half the corneal circumference, lies from one end to the other in the same plane the limbus, and comprehends at its tip a small piece of the conjunctiva. It can be done in one onward and backward movement of the knife, but mostly some sawing is necessary to complete the section.

Second Step: Opening (Peripheric Incision) of the Capsule.—The operator gives the knife back to the assistant and takes from him a cystotome, which he introduces into the anterior chamber, with the knee forward, from the temporal side, near the conjunctival flap, which latter he is careful not to drag into the eye; he then advances the instrument so that the tip goes underneath the upper part of the iris, turns it, and with the tooth makes the incision into the upper part of the capsule parallel with the corneal section about six or seven millimetres in extent. As soon as the capsule is opened the lens makes a visible forward motion. Then the cystotome is withdrawn again with the knee forward, so that the point does not injure the iris.

Third Step: Expulsion of the Lens and Remnants of Cataract.—After the fixing forceps are removed, the operator presses with a Daviel spoon on the lower part of the cornea directly towards the centre of the globe. This makes the flap gape; the lens pushes the upper part of the iris up, tilts the corneal flap forward, presents itself in the gap, and under continued pressure, always towards the centre of the globe, it will slowly rise to make its escape. When its bulk has passed the pupil,—but not before

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that,—the operator follows the lens up and tries by slight stroking and pressing movements to expel the lens together with all the cortex.

The speculum is now removed; the eye is closed, and allowed to rest a minute.

Then the *pupil is cleansed*, if remnants are left, by pressing the edge of the lower lid over the cornea from below upward, while the operator with the other hand keeps the upper lid raised. In this way the remnants, together with the iris, are pushed between and beyond the edges of the wound. This manœuvre frequently has to be repeated several times, and great care should be taken lest, during all this handling, the edges of the lids in any way touch the raw surfaces of the wound. The remnants should be stroked out of the wound with a spatula taken directly out of the antiseptic, for nothing is more apt to carry bacteria into the wound than—what was formerly very common—wiping the wound clean with the edge of the upper lid. Still more objectionable, however, is it to use a Daviel spoon, with which the lens has just been expelled, to fish remnants out of the anterior chamber. Any instrument that is introduced into the eye must be perfectly aseptic, and not have touched the cornea or anything that might harbor micro-organisms.

Fourth Step: Adjustment, Cleansing, and Dressing of the Wound.—During the operation a few drops of antiseptic should from time to time be dropped upon the wound and the cornea. If the iris recedes and takes its natural place spontaneously, any *débris* that may still lie in the wound are stroked out and the conjunctival flap is adjusted with a spatula.

If in the adjustment of the wound the iris does not return spontaneously into its natural position, we may frequently make it do so by pressing upon the lower portion of the cornea, thus causing the wound to gape, which will disentangle the iris when it is crowded in the wound or in the sinus of the anterior chamber. Should this not be sufficient, we have to reduce the iris with a spatula or a blunt probe. If larger portions are within the wound, they can easily be pushed into the peripheric portion of the anterior chamber with the spatula, where they lie crowded in the iris angle. To dislodge them from there a blunt-pointed probe, appropriately bent, should be introduced under the opaque border of the anterior chamber, and the iris unfolded with gentle stroking. If the pupil should be only partially round, we have to repeat the manœuvre and pass the tip of the probe under each point of the opaque corneal border towards which the pupil is still drawn. We should not desist from our attempts until the pupil is central and perfectly round; but if this is either impossible or requires too much manipulation, a portion of the iris must be excised.

When the lens is expelled we recognize by the visual test whether remnants are left or not. The patient should be made to count fingers before his eyes. In case he cannot do that, though by ordinary daylight the pupil appears sufficiently black, the best thing is to examine it with artificial light. If the artificial light should reveal no obstruction in the

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pupil sufficient to account for the bad vision, it is better to abstain from further cleansing manœuvres. But if, as is mostly the case, we find some remnants, we must use our judgment whether we had better leave them alone or continue the cleansing manœuvres. Syringing or scooping them out with a Daviel spoon is often inefficient, and not always harmless.

The conjunctival sac is washed out with a few drops of an antiseptic, the lids are closed, and a patch of sterilized gauze dipped in a weak solution of corrosive sublimate is placed carefully on the eyelids. Over this a pad of moistened cotton is put so as to form a well-adapted cover (a splint), which, by filling the depressions around the eyeball, lies upon the eye without exerting the least pressure. This may be held by a roller bandage or by strips of isinglass plaster, as has been described. To prevent the patient from opening the wound accidentally or by rubbing the eye with the hand, various contrivances are used,—wire or stiff-cloth masks, plates of aluminium, etc. Of the different covers I have found *curved mica plates*, such as are used to prevent foreign bodies from striking the eye, the best. They are fastened by court-plaster strips. They are light and transparent, and not so apt to steam as glass. I have used them for years. Tying the hands so that the patient cannot during sleep or on awaking touch the eye is efficient, and not unpleasant to most patients.

MISTAKES AND ACCIDENTS DURING THE PERFORMANCE OF THE OPERATION.

When the knife passes through the anterior chamber its point is apt to engage in the iris. Farther on, if it has escaped the iris, the *counterpuncture may not be exactly at the point desired.* Both accidents are easily corrected by withdrawing the knife a short distance to disengage the iris or to correct the false position at the counterpuncture.

It may happen that the operator *has introduced the knife with the back up instead of with the edge*, and becomes aware of the fact only when, in continuing the section, the knife does not cut. The mistake can be remedied, without interrupting the operation or injuring the eye, by simply turning the knife at an angle of one hundred and eighty degrees on its longitudinal axis and continuing the section as if the accident had not occurred. In about three thousand extractions this accident has happened to me four times; others have also mentioned it. There has never been any reaction seen from it.

A frequent accident is the *falling of the iris over the knife on cutting upward;* this is mostly caused by an untoward movement or pressure on the part of the patient. It can frequently be made harmless by turning the edge of the knife slightly forward in continuing the section. If, in spite of this, the iris remains before the blade, it has to be cut and the operation finished in the ordinary way. The consequences of this accident are commonly no more than the formation of an artificial pupil, which, if small, will not interfere with the escape of the lens through the regular

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pupil; but if large, and the sphincter portion remains undivided, the latter should be excised before the capsule is opened.

If the *knife deviates in any way from the plane of the section*, it will make an *uneven surface* and the *apex may lie either too far in front* or *too far back*. An uneven section is less favorable for primary union. If the apex falls within the cornea the section closes less accurately, and is apt to cause what is called "a riding flap," which, having a smaller surface of contact between the lips of the wound, does not present favorable conditions for agglutination, and, on the other hand, leads to a kind of gutter between the anterior and posterior lips of the wound, constituting an element of danger in so far as it favors the occurrence of secondary wound infection. To counteract it an exactly applied pressure-bandage should be used and the patient urged to keep as quiet as possible. If the apex of the flap *lies too far backward* it entails an inconveniently large conjunctival flap and invites prolapsus of the iris. Should the latter condition show itself very markedly during the operation, the best thing to do is to make a small iridectomy at once.

In opening the capsule with the cystotome we may press the lens too hard, so that the zonule of Zinn is ruptured. This easily happens in hypermature cataracts where the capsule is thick and tough and the suspensory ligament of the lens frail. As soon as the operator notices it he should stop and either remove the lens within its capsule or try to let the outgoing lens enlarge the insufficient capsular opening. Should, however, difficulties arise, iridectomy ought to be made. In such cases as tremulous or hypermature cataracts in general, where prolapsus of the vitreous is to be dreaded, it is best to remove the speculum and expel the lens by external manipulation. This is done by letting the patient gently look down and pressing the edge of the lower lid over the cornea, while the upper lid is held back with the other hand, and the pressure so directed as we do with the spoon,—*i.e.*, pressing at the lower part of the cornea to make the lens tilt, and, in escaping, block up the wound so that the lens comes out either without or with but little vitreous humor.

Sometimes it happens that we *fail to open the capsule* without being aware of it. This will be recognized when we see that the lens presents in the wound, pushing the upper part of the iris forward, but on continued pressure does not advance beyond a certain point. The remedy is to reintroduce the cystotome and be careful that this time the capsule is surely opened.

If during the expulsion of the lens, when the capsule is well opened, the lens does not come out, the obstacle, in almost every case, will be found to lie *in an insufficient corneal section*. Instead of making attempts to squeeze the lens through, which at best would strip off the cortex, it is better at once to dilate the wound with a fine but strong curved pair of scissors. The best pattern for this purpose are the strabismus-scissors which George T. Stevens has devised for insufficiency operations. (Fig. 11.)

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In a certain number of cases the *obstacle to the exit of the lens consists in a narrow, rigid pupil.* Before resorting to an excision of a piece of the iris, we may try to push the latter back over the lens with the wire loop, and in the majority of cases we shall succeed without damaging the eye in any way.

An *insufficient section of the cornea is the worst mistake the operator could make,* according to the testimony of many earlier and modern authors, with which the present writer entirely agrees. In an insufficient section the iris and the lips of the wound are bruised, the expulsion of the lens is laborious and mostly incomplete, and prolapse of the vitreous may be caused, all of which are favorable conditions for wound infection and destruction of the eye.

Prolapse of the vitreous is one of the common accidents in cataract extraction. It may be unavoidable or avoidable. Unavoidable we may consider it in those cases where the eye is diseased, in particular the suspensory ligament of the lens frail or ruptured (*cataracta tremulans*), or in those where, for some reason or other, we want to remove the lens within its capsule; yet even then we may by judicious handling of the eye avoid prolapse in a considerable minority of the cases. When prolapse is either threatening or has actually occurred before the escape of the lens, we ought to remove the speculum and expel the lens by external manipulation. Experience in that line has taught me how efficient this manœuvre may be under apparently very unfavorable conditions. If the lens sinks into the vitreous, and cannot be removed by external pressure, we must resort to a traction instrument,—the wire loop (Fig. 15) is the best,—with which the lens is drawn out.

Prolapse of the vitreous occasionally occurs during the expulsion of remnants of cataracts, if the operator in the attempt thoroughly to cleanse the pupil presses too hard on the eyeball. We need our best judgment in these cases, being aware that some remnants left behind are less dangerous than the protrusion of vitreous, especially if this occurs in addition to a good deal of manipulation, and above all to the bruising resulting from an insufficient section. We should further bear in mind that there are opacities in or on the capsule which nothing short of extraction can remove. As a rule, we may try to expel the opacities that can be moved with rubbing, but we should leave those in the eye that do not distinctly change their position.

Hemorrhage from the iris is not rare. It may render the operation difficult, but has no bad consequences.

A strange, rare occurrence is the *version of the lens*, by which the upper margin of the lens, instead of escaping through the section, turns downward through the anterior chamber, until the lens has made a rotation of one hundred and eighty degrees, and escapes with the lower margin first, a perfect *breech delivery.* I have seen this four or five times; no accident occurred, and the recovery was uneventful.

Incomplete evacuation of the cataract is an unfavorable accident. It is not always to be avoided. The posterior cortex is particularly difficult to

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remove. We should not push the cleansing manœuvres too far, lest prolapse of the vitreous ensue, or a septic condition in the wound be brought about by coarse and long-continued rubbing over the wound.

The saddest accident, fortunately one of the very rarest, is *abundant hemorrhage from the depth of the eye.* This may occur during the operation or very soon after, and manifests itself by intense pain and the appearance of a stream of blood from the wound, or later by a large clot protruding through the gaping wound.

Dr. J. A. Spalding[1] has found fifty cases of choroidal hemorrhage reported in literature. In a number of cases where choroidal hemorrhage followed extraction in one eye the same accident occurred in the other; but in some instances the second eye remained free from it. The treatment is to apply a compress bandage. Although the eyes became blind, in some cases the shape of the eye was saved; in the majority the globe was destroyed by suppuration.

After-Treatment.—If the patient is restless, has a cough, or unusual pain in his eye, an anodyne should be given. The usual reaction is pain for about six hours after the operation. It is advisable to inspect the wound the next day, even if the patient feels no discomfort. The light should be held by the assistant to the temporal side of the patient in the prolongation of the palpebral fissure. If the bandage is dry, we may infer that the wound is closed. The eyelids should be gently washed with sterilized cotton, the lids parted, and the cornea, pupil, and wound cautiously exposed and illuminated with the lens to the temporal side for inspection. If the pupil is round and central, a drop of atropine may be instilled; if it is distorted upward, there is either incarceration or protrusion of the iris. In the latter case it is best to cocainize the eye, insert a speculum, let an assistant steady the globe with the fixing forceps, as usual, and under focal illumination excise the displaced iris at once. In nervous persons this operation should be done under general anæsthesia. After the excision it is important to push out of the wound any iris that may still lie between its lips. The wound should look just as clean as if a regular iridectomy had been done during the operation.

If the iris is incarcerated without protruding, in the majority of cases there will be smooth healing, but in any case more astigmatism, and sometimes reaction and subsequent prolapse, may occur,—chances to which the operator should not expose his patient, but make an iridectomy as soon as he notices that the incarceration, instead of diminishing, is increasing.

The wound may be inspected daily, but after the second day it is not necessary to expose the eyeball and the wound unless there be some disturbance; after the fourth day in the good cases the wound need no longer be dressed, but during five days more it had better be covered with a patch of sterilized gauze fastened with a strip of court-plaster above the brow. The

[1] Archives of Ophthalmology, 1896, p. 92.

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patient should be warned not to use his eyes except when eating and in other necessary occupations, but not for the purposes of conversation. It is not altogether superfluous to forbid the patient to make any attempts to try whether he sees with the eye operated upon. On the fourth or fifth day, if everything goes well, the patient may be allowed to sit up for an hour or two. The patient need not have his bowels artificially relieved for three or more days, if he does not feel uncomfortable, and he should be warned, on calls of nature, not to strain. In the good cases the patients may be discharged from the hospital in the third week after the operation. It is, however, not prudent to let them travel immediately. An eye operated upon for cataract remains sensitive for at least six weeks, and the patient ought to be most careful during that time. It is advisable to let him wear smoked protectors, and he should not be subjected to an ophthalmoscopic examination or prolonged visual tests before his discharge.

REACTIVE PROCESSES.

I. *Tardy closure of the wound* occurs in a number of cases without any assignable cause, but I have a suspicion that if we could inspect the wound sufficiently we should in some cases find particles of tissue, such as conjunctiva, lens, or iris, between the lips of the wound. As it is a well-known fact that eyes with tardy closure do not suppurate, we need not look upon this condition with apprehension. We merely have to wait about a week —sometimes longer—to see the wound close without any detriment to the position of the pupil or the condition of the eye. It is, however, not true that in every case the tardy closure prevents prolapse of the iris, as Parinaud maintains. The treatment in those cases consists in keeping the eye longer and more carefully closed than usual.

II. *Hemorrhage* after as well as during the operation is rare in simple extraction, and when it occurs it is almost exclusively traumatic; usually the place where the wound was touched can be recognized by a small coagulum. In some cases, not the majority, the occurrence of hemorrhage, by spontaneous as well as traumatic or other bursting of the wound, is followed by incarceration or protrusion of the iris. Its occurrence is indicated by a sudden pain which lasts from half an hour to an hour. If the patient, voluntarily or upon being questioned, in this direction, reports the sudden onset of such pain, the wound ought to be inspected at once and treated according to the condition. We should, however, not omit to inspect the wound on the second day, and every day after, as mentioned above, for in some cases we may be surprised by the presence of a small prolapse which did not cause any discomfort to the patient.

III. *Filtration chemosis* is an innocent condition, though it may look grave. It presents itself as a glossy œdema, mostly on one side and the lower part of the conjunctiva, according to the position of the patient, whereas the opposite side is not swollen. The anterior chamber may be restored or more or less empty. That this œdema comes, however, from

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a leak in the wound is proved by the fact that it always gravitates and causes no pain or other irritation. Its treatment, just as in the tardy closure of the wound, should be a carefully applied compressive bandage, which, while keeping the eyeball immobile as much as possible, does not press upon the cornea.

IV. *Striped keratitis* is a peculiar opacity which shows a number of parallel gray lines vertical to the section of the cornea nearest to the wound; it occurs in sections the apex of which lies at some distance from the limbus and where expulsion of the lens required considerable effort and manipulation. Striped keratitis is not an unusual symptom, almost always disappears, and, according to C. Hess, is produced by wrinkling of the deeper layers of the flap. The treatment consists in careful bandaging.

V. By the end of the second week we notice, in a small number of cases, a very peculiar condition, namely, *herpes corneæ*, also *filamentous keratitis*, in an eye which thus far had shown no reaction. There are sudden pain, lacrymation, and mild circumcorneal injection, and when we inspect the eye soon after the onset of these symptoms we find on the surface of the cornea about a dozen small vesicles out of each of which hangs a small tissue-thread. Within from six to twenty-four hours the herpetic efflorescences will have disappeared, and the patient feels comfortable, but there are relapses of these outbreaks either every day or every few days, which in about two or three weeks disappear without damage to the cornea or eyeball. In some cases such small threads also hang out of the wound. Microscopic examination has shown them to consist of long-drawn, twisted epithelial cells. Instillations of mild antiseptics, careful washing, and a pressure bandage have, in the cases that came under my notice, invariably produced perfect recovery.

VI. *Simple Posterior Synechiæ.*—It is common to see a number of filiform synechiæ uniting the edge of the pupil to the lens capsule, and in the great majority of cases the synechiæ are agglutinations of two raw surfaces, namely, shreds of capsule with lacerations in the pupillary edge of the iris. The proof of this, what I might call mechanical agglutination, is that in the extraction of the lens within the capsule it never occurs, and in peripheric incision of the capsule almost exclusively at the capsular wound. I have been able to demonstrate this in hundreds of cases; there were synechiæ only where the iris touched the capsular incision, whereas everywhere else the pupil was free; moreover, when the incision of the capsule was made sufficiently near the equator of the lens, so that the pupillary border could not get in contact with it, there was no posterior synechia. This is the chief reason that has determined me to prefer the peripheric incision to the central, especially to the promiscuous laceration of the capsule. In every case of simple extraction of a hard cataract we can notice that the lens during its exit lacerates the pupillary edge of the iris in many points, although after the expulsion and the reduction of the iris we see nothing more of it.

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In order to prevent synechiæ it is wise to put a drop or two of atropine into the eye the second or third day after the operation, and keep up the instillations for a week or longer.

VII. *Spongy (or Gelatinous) Exudation in the Anterior Chamber.*—This remarkable occurrence begins with alarming symptoms. The patients have considerable pain, eyelids and conjunctiva are red and swollen, the pupil is narrow and immovable, the iris dull, and the anterior chamber filled with a turbid exudation having the appearance of a fine cobweb; sight is very much impaired. In a few days some part, mostly the upper, of the anterior chamber clears up, and the exudation shows a sharp convex edge, not unlike the crystalline lens. From day to day the exudation becomes smaller by contraction and absorption, and as soon as it disappears from the pupil good sight is restored. Commonly it disappears altogether in less than a week, without leaving a trace. The iris is bright and the pupil black again. No treatment is necessary for this condition, which, in the course of years, I have noticed dozens of times after cataract operations, where it presents the same picture as it does in gonorrhœa and syphilis.

VIII. From the simple, non-inflammatory synechiæ to *true iritis* there is but one step. Iritis manifests itself usually by nightly exacerbations of pain, circumcorneal injection, chemosis, dulness and discoloration of the iris tissue, etc. It is more plastic than serous, producing numerous posterior synechiæ with incomplete or complete closure of the pupil. It should be treated as a genuine iritis with leeches, atropine, and aperients; it may last from several weeks to several months. It is quite rare in simple extraction with peripheric division of the capsule. Rheumatic constitutions predispose to it.

IX. *Irido-cyclitis* is only an extended and graver form of iritis. It is apt to lead to distortion and closure of the pupil, sometimes also to plastic formations in the vitreous with detachment of the retina and shrinkage of the eyeball. Perception of light may be preserved in cases of moderate intensity, and a subsequent iridectomy or, better still, irido-cystectomy may restore useful vision. In the majority of cases, however, especially when the shape and tension of the eyeball are diminished, operations will prove a failure.

Irido-cyclitis should be treated strictly antiphlogistically, the same as the severer cases of iritis.

Irido-cyclitis may extend over a number of months with alternating aggravations and remissions accompanied by corresponding alternations of increase and diminution of eyeball tension. The iris becomes dull, discolored, and uneven. The pupil is completely closed and sight reduced to mere perception of light. This condition in a certain percentage of cases leads to the destruction of the other eye by *sympathetic ophthalmitis*. The latter disease has a very insidious course, beginning with the faintest circumcorneal injection, dulness of the iris, and exceedingly fine, filiform synechiæ all around the pupil. As soon as in the operated eye the symp-

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toms of irido-cyclitis, especially alternations of increase and diminution of tension, are declared, and no hope for restoration of useful sight can be entertained, the eye should be enucleated, without waiting for the appearance of any symptoms of sympathetic inflammation in the other.

Sympathetic ophthalmia has occurred four times in my practice (about three thousand extractions), twice after Graefe's peripheric linear, and twice after simple extraction; in all the iris had been cut, in the former during the extraction, in the latter after the iris had protruded. From this it follows that *sympathetic ophthalmia has occurred in one case out of seven hundred and fifty.*

X. *Cyclitis.*—In a number of cases where the operation was without accident, and the recovery undisturbed during the first week, we notice in the second week deep-seated circumcorneal injection with preservation of a round, movable pupil and good sight, but great pain, especially at night. This may last about a week, when we notice that the capsule becomes dull and thickened, and gradually posterior synechiæ make their appearance. In four or five weeks the disease may have run its course without having done the eye much damage. In other cases, however, there will be circular synechia and the sight will be impaired by wrinkling and dotting of the capsule. In the latter cases, when the capsule is not thickened nor the pupil occupied by inflammatory processes, a subsequent operation (discission) mostly suffices to restore good sight. Another course, however, of those cases of cyclitis is towards development of glaucoma. The eye remains painful, gradually gets hard, and, under continuance of the irritative symptoms, loses its sight. The treatment of cyclitis should be atropine, leeching, salicylate of sodium, anodynes, and, perhaps, alteratives. It should never be omitted in such cases to test the eyeball tension daily, and as soon as there is a decided increase which is not permanently removed by two per cent. pilocarpine instillations, iridectomy should be done.

XI. *Transient choroidal and retinal solution* is a very rare but marked condition. Haab considers it to be due to retinal cysts. In a pronounced case of my own practice a patient with very inflammatory symptoms showed, in the third week after the extraction, a defect in the upper part of the visual field, and corresponding to it a dull grayish opacity in the lower half of the background of the eye. The patient was discharged in the fourth week, and in the course of two or three months had regained a complete field of vision with very good sight.

XII. *Partial and total suppuration of the cornea,* formerly of tolerably frequent occurrence, now reduced to about one per cent., is the result of infection chiefly by infective conjunctivitis caused by dacryocystoblennorrhœa, which mostly starts in the first night, with more or less violent pain, aggravated the next day, running of hot tears, afterwards purulent discharge. When we open the eye the next day we find the dressing moist and more or less impregnated with pus, the edges of the lids red, glossy, swollen, and the conjunctiva red and swollen all around, the cornea dull, and the

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edges of the wound showing a whitish infiltration. These inflammatory symptoms may in a day or two diminish in intensity, the pupil and iris become clearer, the edges of the wound cleanse themselves and agglutinate, and the whole inflammation be substantially over in a week or two (*partial suppuration*).

In another group of cases the inflammatory symptoms will aggravate, the cornea be more opaque, the anterior chamber turbid, making the iris and pupil appear dull; the purulent infiltration of the corneal flap will become more intense, and for about a week or two the symptoms may keep up and then gradually abate; the cornea in its lower two-thirds may clear up; the iris, however, will be united to an indrawn scar with which the cicatrization of the wound is terminated; perception of light may be good, and by iridectomy or iridotomy in certain cases a moderately useful sight may be restored.

In other cases, again, the suppurative process destroys the whole cornea and a *flat leucoma* with perception of light is the issue. In the most intense cases, however, the infiltration of the wound which we noticed on the second day will produce, under aggravating symptoms, a yellowish-gray, ring-shaped infiltration around the entire periphery of the cornea, the well-known *ring abscess*. These cases without exception terminate in total sloughing of the cornea, and almost always with extension of the suppurative process to the deeper structures of the eye (*panophthalmitis*) the eyeball shrinks, but will remain free from irritation, without jeopardizing the integrity of the fellow-eye.

Suppuration may also begin in the iris and the vitreous; the cornea and even the section may be fairly clear, the wound closed, but the iris under symptoms of great irritation may become discolored, and the pupil dull, with puriform exudation in its area and at the bottom of the anterior chamber (*suppuration of the iris*). Under aggravated symptoms the eye will protrude, the corneal wound will burst, and the eye be lost under the picture of panophthalmitis.

Not very rarely, *in cases of prolapse of the vitreous* which seem to run a perfectly smooth course during the first four or five days, we notice under grave inflammatory symptoms a dullness of the pupil and a yellowish reflex from the vitreous chamber (*suppuration in the vitreous*); vision is gradually lost, the eye will swell, and panophthalmitis will develop.

The *treatment of all these suppurative processes* in the great majority of cases is very unsatisfactory; it is only in the milder cases of partial suppuration of the flap that the eyeball may be saved; and if it is saved, so far as my experience goes, it is owing more to a low degree of virulence of the infective material and to a strong vitality of the subject than to any one of the many remedies used and praised for this condition. I have not found that galvano-cautery or disinfectants of the wound are particularly beneficial. Opening of the wound and drainage of the anterior chamber, repeated every day, had more effect than any other means I have tried.

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VARIETIES OF THE OPERATION.

Extraction combined with iridectomy is the method which rivals the flap extraction originated by Daviel, and at the present time seems still to boast of the greater number of followers. It consists of a section exactly the same as above described; a small excision of the iris is made and the operation continued in substantially the same way as in the simple extraction. It is asserted that in the expulsion of the lens the spoon need not be pressed directly towards the centre of the globe, but may follow the lens in its exit from beginning to end.

The *opening of the capsule* is done in various ways by different operators; mostly it consists in an *extensive and promiscuous laceration.*

A number of operators are in the habit of *removing a portion of the anterior capsule*, especially when it is thickened, which is done with the so-called *capsule-forceps*, like Mathieu's, and a number of other patterns, all of which have the essential feature that *the teeth are on the lower surface instead of at the point.* If this manœuvre succeeds, it mostly leaves a clear space in the centre of the pupil. If it is applied to thin capsules, it does not differ essentially from the promiscuous laceration. In thickened capsules it is not uncommon that *the whole crystalline lens together with the unopened capsule is pulled out.* If the centre of a somewhat thickened capsule is pulled out, it occasionally happens that the *suspensory ligament is ruptured*, and the expulsion of the lens is complicated with prolapse of the vitreous.

An old method of opening the capsule, revived by Gayet, Galezowski, and others, and which I have also practised in a continuous series of over twenty-five cases, is to *split the capsule with the knife on its passage through the anterior chamber.* I abandoned it because the opening was insufficient in many cases, and it rendered an exact execution of corneal section more difficult.

The *removal of the lens with an unbroken capsule* is an old method revived by Alexander Pagenstecher and his brother Herman. The section is followed by an iridectomy, and the lens is drawn out with a large spoon introduced along its posterior capsule. In a very great number of cases the operation is followed by *prolapse of the vitreous.* It has not been found practicable to introduce this mode of extraction as a general method, for in the attempt to extract the lens in by far the greater number of cataracts the capsule bursts. The *method is now limited*—even by its warmest advocates—*to hypermature, tremulous*, and *dislocated cataracts.* With this restriction, the present writer finds the method perfectly legitimate and advantageous, but as to its execution he prefers to expel the lens by external manipulation if possible, which is mostly the case. He is not aware that for the last fifteen years he has made use of any traction instrument, and yet he has left the cataract in the eye in only two very complicated cases.

Linear extraction consists in making an opening into the cornea about a line from its periphery by a lance or a knife. The capsule is then lacerated

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and the lens expelled, either with or without previous iridectomy, by pressing the posterior lip of the wound back with a spoon gently on the opposite side of the cornea. The operation is adapted exclusively for soft cataracts, and the opening of the cornea may be made either on the outer or on the upper or lower part of the cornea.

Jacobson's *combined peripheric*, v. Graefe's *combined peripheric linear*, and Schuft-Bowman-Critchett's *scoop extractions* have deservedly been abandoned. They did not, as was claimed, materially prevent suppuration, but engendered an inordinate number of cases of irido-cyclitis followed by phthisis bulbi and not exceptionally sympathethic ophthalmia.

Removal of cataract by suction has been practised for about fifty years. It has found its advocates, but has never gained a hold on the profession. It is applicable only to cases of soft cataract, and in this way is intended to replace linear extraction.

With a broad needle or a Graefe knife an opening is made into the cornea about three or four millimetres from its margin, and then also into the lens capsule. Into the corneal opening the nozzle of an aspirator is introduced, the tip of which is pushed into the pupillary space and the lens matter sucked out either by the mouth or by some artificial suction arrangement.

The nozzle is a flattened, slightly curved canula, having on its concave side an oval opening: its base is connected with a glass tube to receive the lens matter; the other end of the glass tube is connected with an india-rubber tube, which at its other end is connected with a mouth-piece (Teale) or a suction-bulb (Bowman and others). At the point of union of the rubber tube with the glass tube is a valve preventing air from entering the anterior chamber.

The operation is to replace or supplement discission of soft cataract. It cures the patient more quickly, but less safely. A good deal of manipulation, requiring particular precaution, may produce inflammation of the iris and vitreous. The few cases which I have operated on by suction produced imperfect results. The evacuation of the cataract was not complete, and posterior synechiæ developed. Suppuration has also been noticed to follow it. The operation has not met with great favor. Coppez and Redard have of late recommended it warmly, particularly for traumatic cataracts.

The *extraction with simultaneous corneal and iris flaps*, according to Wenzel, refers to cases of total posterior synechia,—*i.e.*, adhesion of the whole posterior surface of the iris to the anterior capsule. It is a matter of frequent experience that in such cases an iridectomy will remove only the anterior layers of the iris, but leave the dense and dark uvea unbroken upon the capsule. Wenzel thrust his cataract-knife as soon as it had pierced the cornea also into the adjacent part of the iris, pushed it straight forward through the lens, pierced the iris and cornea on the nasal side, and completed the section as in an ordinary flap extraction, thus forming a corneal and an iris-capsule flap. Of the latter he excised as much as he could, and then extracted the lens. It is an operation applied to desperate cases, where a moderate visual result would be a surgical triumph.

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III. DISCISSION.

Discission is indicated:

(1) In soft cataracts of young people, up to the age of fifteen, as is generally alleged; but before the reintroduction of simple extraction I have successfully and with the best visual results removed cataracts by discission up to the age of thirty-seven, in patients whose eyes I did not want to mutilate by the broad iridectomy then fashionable.

(2) To *ripen* cataracts. See page 795.

(3) As a preparatory *step in the surgical treatment of high degrees of myopia* (15 D. and over) by the extraction of the lens. This operation, revived by Fukala, is at present frequently performed in Europe, especially in Germany. In America there is not so great an opportunity to operate on excessive degrees of myopia; apart from that, I confess not to be very partial to removing a non-cataractous lens. There are many successful cases reported, but I do not doubt that there will also be cases in which the lenses could not be removed cleanly and the results were unsatisfactory, to say nothing of losses by suppuration, irido-cyclitis, intra-ocular hemorrhage, and subsequent detachment of the retina. The proper value of this treatment of myopia will be recognized when the period of its "boom" is passed.

(4) As the most frequent operation for *secondary cataract*, when a wrinkled or dotted capsule, remnants of lens enclosed between the two leaves of the capsule, or other not markedly inflammatory deposits obscure the pupillary space. If the sight is good, 20/40 or more, discission may be omitted. If the sight is not so good, we must ascertain whether the capsule is the cause. If we look into the eye with the ophthalmoscope and get a clear image of the background, discission is not indicated.

Instruments required.—Speculum, fixing forceps, operating chair, artificial light, etc., as in extraction. Further, discission needles, of which for the different cases three sizes are indispensable. Needles of all forms have long been in use,—curved, straight, sharp on one or both sides. After trying all of them, I have for years given preference to a so-called *knife-needle* (see Fig. 16), cutting on one side only, the blade and the evenly round shaft so proportioned that the shaft fills exactly the opening made by the blade, so that the needle can be moved within the anterior chamber in every direction without escape of aqueous on the one hand or bruising of the cornea on the other. The lengths of the blades are (1) three millimetres (small size), for discission of the anterior capsule in soft cataract; (2) four and a half millimetres (medium size), for the majority of secondary cataracts; (3) six millimetres (large size), for larger openings of the capsule and deeper breaking up of soft cataracts or transparent lenses in young myopes of high degree, as Fukala recently recommends.[1]

[1] Heilung höchstgradiger Kurzsichtigkeit, Vienna and Leipzig, 1896, p. 48, and Arch. of Ophthalmol., vol. xxvi. p. 174 (book review).

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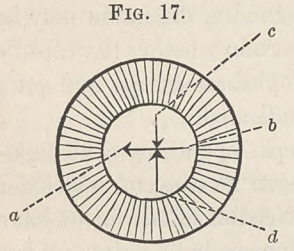
I prefer the straight knife-needle, because needles cutting on both sides can for equal sizes not be made so sharp as straight ones, and curved needles are difficult to introduce through the cornea and still more so through the capsule. The needle has to go two and mostly three times through a thin, elastic membrane, which readily escapes the instrument. The straight point transfixes the membrane with greater ease, less pressure, and, therefore, less tearing at the ciliary processes. The knife-needles should be of the utmost sharpness both in point and in edge, for they are intended to cut and not to tear. They should be very carefully handled in cleaning, so that the point is not bent and the blade not dulled by cutting against the linen used in wiping them. They wear out by sharpening more quickly than other instruments. The hone which takes off a little of the width of the blade destroys also the proportion between the size of the blade and the thickness of the stem.

Performance of Discission.—For primary soft cataract we select the small-size knife-needle, and thrust it through the artificially well-lighted cornea three millimetres from its margin in the horizontal meridian. The needle is advanced to from one and a half to two millimetres beyond the anterior pole of the lens, thrust through the lens capsule, and drawn back temporarily through the capsule and the superficial adjacent layers of the lens, so as to make a horizontal incision of four or five millimetres. (See *b*, *a*, Fig. 17.) Then the point of the needle is raised towards the cornea, and pushed upward in front of the capsule, which it transfixes (*c*, Fig. 17) from two to two and a half millimetres above the horizontal incision, and divides it downward as far as the horizontal incision. The same manœuvre is done on the lower half of the capsule from below (*d*, Fig. 17) upward, so that the three cuts make a crucial opening into the anterior capsule and the adjacent layers of the lens of from four to five millimetres in length respectively. If the lens substance is somewhat coherent, sticky, the needle may be turned in the opening, and a flake of lens substance lifted into the anterior chamber. The needle is then withdrawn quickly through the cornea, so as to prevent the aqueous and particles of lens from escaping or becoming caught within the little canal.

Fig. 17.



Incisions with the knife-needle in soft primary and in secondary cataract.

For *ripening* of a cataract and making *the transparent lens of young, excessive myopes cataractous* and fit either to be absorbed or extracted, the technic of discission is essentially the same as in primary soft cataract, only that the incisions may be longer and deeper.

For *membranous secondary cataracts* the technic is fundamentally the same as in soft primary cataract, but the greatly varying character of the capsule or pupillary membrane requires a great many deviations from the general plan. Each case has to be studied for itself, and the incisions have

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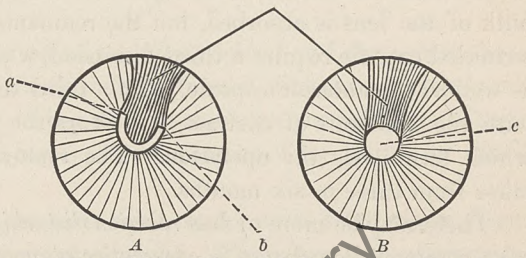
to go through the softest parts of the capsule; hard and inelastic bands and patches should not be attacked, and in case we err in our judgment, finding that such and such a band offers too great a resistance to be severed, we have to cut above and below, to the right or left, as the case may be, creating free pupillary space sufficient for good sight.

Varieties of the Technic.—In the discission for primary as well as for secondary cataract two incisions may suffice, in the shape of a T, or the one crossing the other at an acute angle. This mode mostly furnishes a sufficiently large free space, but not so extended as the crucial division. A single cut should be avoided, as it almost always gives an insufficient opening. In one particular condition a single incision may give an excellent pupil,—namely, when a thin inflammatory pupillary membrane adheres to the iris by circular or filiform synechiæ. The knife-needle, after transfixing the pupillary membrane at a thin place, being swept around the capsular obstruction along the pupillary edge of the iris (see Fig. 18, *A*), detaches the pseudo-membrane from the iris, and by contraction of both pseudo-membrane and iris creates a clear space between these two structures. (See *c*, Fig. 18, *B*.)

FIG. 18.



A, course of the needle swept along the pupillary border in a pupil obstructed by an inflammatory membrane. The curved line *ab* should touch the pupillary edge.

B, *c*, free pupil by the retraction of the pseudo-membrane detached from the pupillary edge.

It may happen that by insufficient illumination or by the reflex covering a portion, usually the lower, of the pupil, *a part of the capsule escapes cutting*, which shows by an insufficient opening in the capsule when the needle is withdrawn. In this case we may introduce the needle a second time in the same sitting, either in the old or in another place. I have done this a number of times without ever seeing any harm from it. We may, of course, desist from immediate repetition of the discission and make the division complete later, say one month or longer.

In tough capsules, either in hypermature cataracts or from iritic products, we may obtain a better opening by a laceration of the thickened capsule with two needles. For this, *the double-needle operation*, I would prefer the small knife-needle likewise, yet the Bowman stop-needle may serve the purpose. The operation is as follows. An assistant steadying the eye with fixing forceps, the operator plunges one needle from the temporal side through the cornea three millimetres from the margin and directly into the centre of the thickened capsule. Then he introduces with the other hand a second needle in the same way from the nasal side into the centre of the capsule, touching the first needle. Now by approaching the handles of the

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needles their blades diverge and tear the capsule in two without pulling at the ciliary processes. The opening thus created is more liable to close again than to remain patent. For this reason a number of the best operators, for instance Panas, are uncompromisingly in favor of extracting all pupillary obstructions that cannot be divided by a discission-needle. Others (Esberg[1] and Schweigger[2]) use reduced forceps-scissors, introduced through a peripheric corneal incision with a lance-shaped knife.

Treatment and Reactive Processes after Discission.—After discission in *primary soft* cataract the patient usually has little or no pain. A drop of a one per cent. solution of atropine is instilled immediately after the operation, and once or oftener every subsequent day, to keep the pupil dilated. The lens substance brought in contact with the aqueous humor will swell, escape through the wound in the capsule particle by particle, fall into the anterior chamber, and be absorbed. When this process has been going on for about six weeks its progress becomes slower and slower, so that a second, bolder, and deeper discission is made to bring new masses under the absorbent influence of the aqueous. After four or five weeks again the bulk of the lens is absorbed, but the remnants adherent to the dotted and wrinkled capsule require a third discission, which comprehends the posterior as well as the anterior capsule. This third discission not only does away with the remnants of cataract, but clears the pupillary space permanently, which terminates the operation. The removal of a cataract in this way takes from three to six months.

There may be more or less *reaction disturbing this favorable course.* The most common disturbance is *consecutive glaucomatous inflammation* from too rapid imbibition of the lens substance. Instead of small particles successively falling into the anterior chamber, the nucleus and even the whole lens may protrude through the capsular opening at once. Great pain, circumcorneal injection, increase of eyeball tension, and loss of the eye by glaucomatous irido-cyclitis may be the consequence. This disastrous course may at once be checked by extraction of the lens with or without an iridectomy. Many operators expect and favor such a course by making a bolder discission from the start, and remove the lens as soon as symptoms of glaucoma are manifest.

Moderate *iritis*, with permanent synechiæ, is a frequent sequel of discission of primary cataract.

Irido-cyclitis is rarer, and suppuration quite exceptional, but all these and other deleterious reactive processes which we have seen to follow extraction may be induced by discission likewise.

The recovery from discission of secondary cataract, as a rule, is excellent. Some pain for from six to twelve hours after the operation is not uncommon. In the majority of cases there is no redness or pain from

[1] Esberg, Zeh. Kl. Mon., 1895, p. 249.
[2] Schweigger, Textbook, 6th ed., 1895, p. 358.

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the next day to the end of the recovery. Most of the patients can safely be discharged on the sixth day, if they live near the operator. I do not like to lose sight of them for the next weeks, as exposures of all kinds are apt to produce irritation, in particular glaucoma.

Among the **disturbances of the recovery** I may mention:

(1) A *film of vitreous protruding through the puncture-canal*, and hanging out like a short, movable, mucoid cord over the cornea. If cut early the irritation caused by it soon disappears, and the recovery is good. In some cases a yellowish gray film leads from the puncture-point into the pupil, indicating the passage-way of the needle. This occurrence, from which I have never seen very grave consequences, is rather rare, but I have noticed and described it for more than twenty years in my successive reports on cataract extraction.

(2) *Reuniting of the capsular opening* is common if only one incision is made; infrequent in the T-shaped division, in which mostly the vertical cut reunites, whereas the horizontal, the one parallel to the opening of the capsule, remains open. A repetition of the division remedies the shortcoming of the first attempt.

(3) *Formed opacities of the vitreous oscillate behind and through the capsular opening.* This occurs in cases where formed vitreous opacities were present before the operation. They impair sight proportionately to their density, and may lead, though rarely, to detachment of the retina, for which the discission is not to blame.

(4) *Irido-choroiditis* may, in rare cases, occur even after many years, and may lead to serious impairment of sight. There are pain, circumcorneal injection, discoloration of the iris, striated parenchymatous opacity of the cornea, posterior synechia, and cloudiness of the vitreous. One such case, setting in six years after a simple extraction with subsequent discission and excellent sight, in a rheumatic young woman, is now under my care. I made an iridectomy; there was no vitreous in the anterior chamber; the pupil dilates fully, and the case is doing well. The worst case I have had was one of glaucomatous irido-cyclitis some months after a discission. The eye was very painful, had great circumcorneal injection, pupil narrow, sight reduced to movements of hand, grumous hypopyon of two millimetres in height, T +1. As soon as the latter symptom appeared I made an iridectomy, and the patient improved instantly. No more pain, the hypopyon disappeared in a day, the pupil cleared up, and in a month her sight was 20/20. This was a recent case, but I had a few in former years, before I was familiar with glaucoma. They all had gone home and exposed themselves by going to work too soon and too assiduously. I readmitted them to the hospital, where they rapidly recovered.

(5) *True suppuration and the loss of eyes from this and other causes* are reported by several authors, but it has been my good luck that no such case in my own practice (except from glaucoma) has come to my notice. I do not think that infection through the discission-needle—the only instrument in-

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troduced into the eye, and which can easily be sterilized—is to be blamed for it, nor the transportation by it of pyogenic germs which may harbor in the region of the puncture, for thus far, in considerably over a thousand cases of discission, I have not seen one of suppuration. The only explanation I can offer is the following. In these cases pyogenic germs had been introduced into the interior of the eye, but in so small a quantity and under such favorable local and general conditions that they produced only a mild reaction, were encapsuled, and lay dormant until a favorable condition—bruising of the cornea, tearing at the ciliary processes, and deep ploughing of the vitreous—waked them up again. Be this hypothesis founded or unfounded, I have, always conscious of the fact, made it my constant endeavor to heal cataract extractions with the least possible traumatism and inflammatory reaction. Considering secondary discission an almost necessary supplement of an extraction whose aim is the permanent restoration of good sight, I cling to the peripheric opening of the capsule and the simple extraction, in order to expose the tissues of the eye to a minimum of wounding, and to avoid numerous iritic and cyclitic reactions which, though not destroying the eye, leave it an injured and susceptible organ, whose tolerance of discission and other hurts is less than that of healthy or but little injured eyes; in other words, *I perform extraction with a view towards the necessity of a subsequent discission.*

(6) *Glaucoma is the only consequence of discission which may be fairly considered as inherent to the operative procedure.* A low degree of increase of tension appears not infrequently, perhaps, during the first twelve hours as reaction from the operation, and disappears without treatment. A higher degree, accompanied by increased pain during the first night, is still manifest the next day by circumcorneal injection, chemosis, and increased hardness of the globe. This degree may likewise disappear spontaneously in a day or two, but it may also develop into well-marked glaucoma, with persistence of pain, swelling and redness of lids and conjunctiva, lacrymation, and impairment of sight. These symptoms increase day by day, and very soon the iris becomes bulging in its middle portion, whereas the ciliary and pupillary edges remain in their normal position. The iris appears somewhat like a tube, wound around the pupil, more elevated in some portions than in others. The media are dull, the fundus veiled. I have only one example in my own experience how these cases terminate without operation. This was a boy, whose right congenital cataract I discinded in his third year. The third division was followed by irido-cyclitis, with gradual though slight dilatation of the ball. An iridectomy stopped the disease at once, but the eye had lost its sight. This was many years ago, when I was ignorant of glaucoma following division of the capsule. Of another case, where glaucomatous irido-choroiditis with hypopyon was cut short by an iridectomy, I have spoken above. There are two cases where glaucoma broke out a week after the discission, when the patients had left New York. Their eyes became blind under the picture of subacute glau-

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coma, with cupped optic disks. The other cases, about twenty in number, were all cured either by myotics or an iridectomy. Perhaps a paracentesis of the anterior chamber may be sufficient, as it was in a case reported by H. Pagenstecher, and another in which I let out vitreous collected in the anterior chamber in an unsuccessful attempt to excise a piece of iris. Thus far I have clung to iridectomy, which has not yet proved a failure. On the peculiarities of the operation in aphakic eyes see page 786. One case, which I discharged on the fifth day with excellent sight five years ago, is now reported to have still S. 20/20, but glaucomatous cupping and contraction of the visual field. Another case with a mild form of glaucomatous cyclitis has now, eighteen months later, presented herself with S. 2/200 and nasal contraction of field. From this I conclude that there may be other cases of glaucoma not brought to my notice, and deduce this rule for guidance, that all cases of never so slight glaucomatous cyclitis should be treated with iridectomy without delay.

RESULTS OF CATARACT OPERATIONS.

Discission of soft primary cataract is a comparatively safe method, yet it has its failures which have not been statistically presented in a large number of cases. Besides irido-cyclitis we have to mention suppuration as the main cause of failure, but the percentage of loss from either seems to be smaller than in extraction. Of extraction we possess extensive statistics, which are chiefly of use for comparing the value of the different operative methods and less important factors in the after-treatment. Unfortunately, the value of statistics is diminished greatly because the basis on which they rest differs very much. In cataract statistics the greatest element of fallacy lies in the judgment what complications, if any, should be excluded from the count. If one operator reports on a series of one hundred cases and excludes thirty cases operated on during these one hundred cases as complicated, and another excludes only thirty complicated cases intercurrent with six hundred, the results are not comparable. The most reliable statistics are those in which every case is counted, and even these have their fallacies. A timid operator who considers preliminary iridectomy not only as "the operation for his grandfather," but as the sheet-anchor for all patients, and shirks unfavorable cases, will have a better statistical exhibit than an operator who, as a rule, practises simple extraction, and does not refuse his services to anybody who has a reasonable chance, however small it may be. It is plain that the exhibits of such men are not comparable. Even if all cases are counted and reported in a table of numerous successive cases, the results can be obtained in different ways, so that the summing up in x per cent. of good results, y per cent. of moderate results, and z per cent. of failures is not equivalent to a mathematical demonstration. This does not mean that I underrate the value of statistics. All statistics are good if they are honest; but they signify no more than what the reader learns by them if they furnish him the data to judge

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each case for himself and estimate the relation to the sum total. Larger statistics from one operator have the value to show what in his experience has stood the test of time, and where progress has been made, be it in the diminution of the number of losses or in elevation of the standard, duration, and percentage of good vision restored. The lack of mutilation can be considered only as a minor factor.

The author may be permitted to draw on his previously reported cases of a series of *1000 successive cases of combined extraction* from 1866 to 1888, published in Graefe's *Archives*, the *Archives of Ophthalmology*, and the *New York Medical Journal*, compiled in a paper published in the *Transactions of the Ophthalmological Society*, 1887.

Considering V. 20/200 to 20/20 as a good result, 18/200 to 1/200 as moderate, mere perception of light and blindness as failure, the visual results were—good, 85.4% ; moderate, 8.3% ; failure, 6.3% ; loss from suppuration, 4.2% ; loss from all other causes, 2.1%. I should not omit to state that cases in which *the cataract was the minor disease*, such as detachment of the retina, etc., were excluded.

From June 10, 1886, to the present day (June, 1897) I have practised the simple extraction as a rule. I have published reports on the first 300 simple extractions.[1] Very complicated cases were excluded. In 37 intercurrent cases iridectomy was made, with 2 failures by suppuration. The 300 simple extractions had an unusually high percentage of success,—namely, good, 96.33% ; moderate, 2.66% ; failure, 1.01%. All the cases are tabulated and described in 3 series of 100 cases each, with critical remarks.

Besides these, I have at my disposal a series of 1000 successive extractions, not yet published, which I have analyzed in two series, the one of 600 cases, with 30 additional cases so complicated that the prognosis was too unfavorable, and the other of 400 recent cases which comprehend all cases that were operated on. There were among these 400 cases 57 with bodily or ocular complications, showing 8 failures,—*i.e.*, 14%. In 56 cases—*i.e.*, 14%—iridectomy was made, with 7 failures, viz., 6 in cases of grave irido-cyclitis, 1 in chronic conjunctivitis. The reactive processes divided into :

A. *Inflammatory.* They showed :

(1) *Mild iritis*, 6 cases. (2) *Protracted irido-cyclitis*, 2 cases. (3) After hemorrhage, 1 case. (4) Slow closure of wound (eleven days), 1 patient eighty-three years old, kept in bed long, contracted pleuro-pneumonia, but recovered ; sight good (20/40 without discission). (5) *Partial purulent wound infection*, 2 cases. (6) *Total suppuration*, 4 cases.

B. *Mechanical.*

(1) *Anterior synechiæ*, a goodly number ; in 1 case severe reaction (glaucoma), iridectomy, recovery.

[1] Arch. of Ophthal., vol. xvii. p. 51 ; vol. xviii. p. 1 ; vol. xix. p. 280.

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(2) *Prolapse of iris*, 26 cases out of 343 cases of simple extraction,—*i.e.*, 7.6%. Result good, except in 1 case, where the prolapse from chronic cough occurred on the third day. Abscission, partial suppuration, good projection.

In 153 cases—*i.e.*, 38%—discission of *secondary cataract* was made: in 4 of them glaucoma developed, which was cured by iridectomy in 3 cases, by myotics in 1. Sight good in all, and materially better than before the discission. Two cases came to my notice later.

The visual result of the 400 cases was: good in 90%; moderate in 7%; failure in 3%.

The *vision* in the series of *the 600 preceding extractions* (30 intercurrent complicated cases excluded) was: good, 95%; moderate, 3%; failure, 2%.

Among the failures there were 2 cases of *sympathetic ophthalmia*, excited by prolapse and its abscission in complicated cases (inveterate rheumatism in one, degeneration of choroid and vitreous in the other). This appalling calamity—2 cases in over 1400 extractions—occurred twice in the series of 1000 cases of combined extraction.

In conclusion, I desire to present the statistics of the *results of the operations for* **secondary cataract**, omitting all but those of discission.

It is generally known that the visual result as determined at the patient's discharge rises somewhat in the next months, but then it gradually sinks in most cases by wrinkling and dotting of the capsule. This goes so far that, to cite an example, a young person operated on by discission and extraction, done by a skilled and most careful English oculist, could see well, read fluently, etc., for four years, then the vision dimmed, and in two years more it was 10/200 in each eye. Discission by crucial cuts restored vision to 20/20 in each eye in ten days, and this vision may be considered permanent. The average result of primary vision after extraction, simple as well as combined, computed from many hundreds of cases, is 20/70, that of ultimate and permanent vision after discission is 20/30. This gain is not, as far as I have experience, counterbalanced by danger inherent to the method, for the 1% of glaucoma is under control by myotics and iridectomy. As to numerical evidence, see my repeated detailed reports.

I have deemed it correct to give in so exhaustive a work as the "System" detailed data of my own experience, supplementing the general remarks made above, to which the reader can refer in parallel cases. I should, however, fail to present a complete picture were I to omit statistics from others.

Daviel made 43 extractions at Reims in 1751. Dr. Caqué, a corresponding member of the Paris Academy of Surgery, reported in 1753 on 34 of these patients as follows: 17, perfect success; 8, moderate success; 9, failure. Daviel himself in his paper to the Academy of Sciences, according to Thomas Hope, 1752, says that of 115 operations 100 had succeeded. Arlt gives 8.80% of loss in flap extraction, 5.67% in Graefe's linear method. David Little in 106 simple extractions had 4 failures, 1

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panophthalmitis, 1 suppuration of cornea, 1 glaucoma, 1 closure of pupil; prolapse in 10 cases, in 17 cases discission of secondary cataract, all good. In 322 cases of combined extraction he had 96% of good, 1.6% of moderate, and 1.8% of failure. In both series all the cases were uncomplicated cataracts. D. Webster in 118 uncomplicated cases had 6 failures. J. E. Weeks in 80 uncomplicated cases, 1 loss by suppuration. Schöler (report by W. Albrand) had of 126 combined extractions 7 failures (5 by suppuration), of 132 simple extractions 6 failures (2 by suppuration). Duke Charles in Bavaria (report by Dr. Zenker), 95.2% good, 3.2% moderate, 1.6% loss (0.9% by suppuration). This excellent report, in which all cases are entered in 10 tables of 100 patients each, includes operative recovery among the good results, even if the pupils were so full of remnants that sight was very poor. All the cases except 5 were operated with iridectomy. Wound infection, 8 after extraction, 1 after discission. Swanzy, in a series of 100 uncomplicated cases, with 30 intercurrent complicated not included, reports in 1890 95 good, 2 partial results, 3 failures. Among the 30 complicated cases there are 7 failures and 9 partial results. The report of the New York Eye and Ear Infirmary for 1896 comprehends 181 patients operated on for cataract (14 complicated), with the following results: V. = 0 in 2.85%, V.= 1/00 in 9.28%, V. < 20/200 in 13.23%, S. = 20/200 or more in 74.64%. Dr. F. M. Wilson, of Bridgeport, reports 100 cases (tabulated), with 5 failures. He took particular pains in following up his cases. In 70, which he succeeded in tracing, 9 eyes had undergone serious changes within three and a half years,—*e.g.*, 1 relapse of irido-capsulitis, followed by *sympathetic ophthalmia*. A *fulminant glaucoma* five months after a successful discission, vision not restored by iridectomy done in twenty-four hours. Another fulminant glaucoma set in eleven days after a successful discission. Iridectomy restored sight, 20/30, in three weeks. Two months after a discission, a patient, during an attack of grippe, had irido-capsulitis with hypopyon; recovered in two weeks. S. = 20/40. Case of discission, three months after extraction, showed in four days a gray, thread-like line passing from the puncture-canal backward. Turbidity of vitreous; capsular wound occluded with gray deposit; hypopyon filling one-third of anterior chamber. Gradual absorption. In three weeks vision raised from 10/200 before the discission to 20/100+. Such reports are very valuable. Dr. Lyman Ware, of Chicago, reports 100 extractions, 12 cases complicated, 4 complete failures, 1 by panophthalmitis, 3 by irido-cyclitis, the others good. Preliminary iridectomy, one week to ten days. Discission in 25% three to six weeks after extraction.

From these and many other reports we may put down the results of cataract extraction, collected from a few weeks to a few years after the operation, at an average as follows: in uncomplicated cases, failure, three per cent.; moderate result, seven per cent.; good result, ninety per cent.; in all cases as they occur, failure, five per cent.; moderate result, ten per cent.; good result, eighty-five per cent.

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If the cases are followed up for more than a few years, we shall arrive at totally different percentages of visual results, which are altogether in favor of the cases in which simple extraction with subsequent discission of the secondary cataract had been performed. Very often such cases have come to my notice from two to eleven years after the operation, with eyes scarcely distinguishable from normal ones, with unobstructed, responsive pupils, clear background, and good and enduring sight. There is no doubt that persons operated on for cataract possess no immunity from any eye-disease, but eyes operated on according to different methods show different degrees of resistance to the deteriorating influences of age, work, weather, and systemic diseases. The eye mutilated by an iridectomy with its frequent contaminations of the corneal scar by never so small or invisible portions of iris, capsule, lens, and inflammatory products withstands these influences less than the eye after simple extraction. Even if we do not forget that in ten per cent. of the cases an iridectomy is indicated for various unfavorable conditions, that further iris prolapse has to be excised in about ten per cent., and that in other ten per cent. there are posterior synechiæ,—*i.e.*, adhesions of iris to the scar after or without a preceding iridectomy,—there remain seventy per cent. of ideal recovery, and the remaining thirty per cent. are much in the same condition as an equal number of combined extractions. From my own experience I have long been forced to the conviction, which the adverse incident literature has not shaken, that *simple extraction is not only the best but also the safest operation for senile and many other cataracts.*

§ IV. OPERATIONS ON THE CORNEA.

I. REMOVAL OF THE SUPERFICIAL LAYERS OF THE CORNEA (ABRASIO CORNEÆ).

This operation consists in the scraping or shaving off of the corneal epithelium and the adjacent strata of the cornea.

It is done with a cataract-knife (Graefe's or Beer's), a lance-shaped knife, or any very sharp and delicate scalpel. Its indications are infrequent, but it seems to me that they might be extended. I have done it:

(1) In *deposits of different substances* on the cornea, especially *deposits of oxide of lead*, which were not rare in former years, when aqua plumbi was a favorite collyrium for all kinds of inflammation.

(2) In the peculiar degeneration of the corneal epithelium, which *opacifies a horizontal strip* of cornea, sometimes in healthy but mostly in chronically diseased eyes, irido-choroiditis and glaucoma. It is known under the name of *chalky film* or *ribbon-shaped keratitis.*

Execution of the Operation.—The eye is anæsthetized with cocaine, eucaine, or holocaine, held firm with an ordinary pair of fixing forceps, or with two of them, or a double-forked ophthalmostat, as great steadiness is required. I have always done these operations tentatively,—namely,

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I removed a portion of the opacity, and, if the result was satisfactory, removed the rest. I scraped or shaved off a part of the opacity from the periphery, but removed all that was in this place, not afraid of the depth of the defect.

When the conjunctiva is aseptic, *clean corneal defects are replaced with no or very little scar.* The recovery has been undisturbed, and vision in some cases greatly improved.

II. INCISION, CURETTING, AND DISINFECTION OF CIRCUMSCRIBED CORNEAL INFILTRATIONS.

Indications.

This mode of treatment has a wide range of application.

(1) *Pustules of the cornea*, round or oval, gray or yellow deposits of inflammatory products. They are single or multiple, produced by foreign bodies, small injuries, or infection from neighboring organs without assignable cause in healthy or dyscrasic, mostly unclean people.

(2) The *marginal*, small, white *infiltrations*, which, if unchecked, coalesce and form the *crescentic, annular, progressive ulcer.*

(3) The *irregular, progressive infiltrations* of the cornea, which, starting in or near the centre, show in different places denser white or yellowish spots, known under the name of dendritic, serpiginous, malarial keratitis.

(4) Other disseminate, punctate infiltrations, evidently of bacterial origin, for instance, tuberculous or trachomatous.

Operative Procedure.—It is best, after thorough local anæsthesia, to incise the infiltrations superficially in case they are prominent or show distinct involvement of the adjacent cornea, to scrape them with a fine sharp spoon, and then disinfect them with tincture of iodine, nitrate of silver, bichloride of mercury, and the like. During the last four years I have given preference to tincture of iodine, which is easy to apply. A bit of absorbent cotton, wound on a probe or a match, is dipped in tincture of iodine, held a few seconds in the air, then pressed on the infiltrated or scraped place either mildly and quickly, or in most cases firmly, and for from three to five seconds. The place will then show a depressed, dark-brown centre, surrounded by slight superficial coloration. As after-treatment I have ordered instillations of boric acid, or more often a 1 to 5000 solution of bichloride of mercury every hour or two. One application may not cure the patient; then the cauterization should be repeated several times.

Our judgment of the extent of the infiltration and the depth of the loss of epithelium and corneal stroma is much facilitated by instilling a *few drops of a two per cent. solution of fluorescine*, which in from two to five minutes will stain green all the denuded and ulcerous infiltrations. As adjuvants, washing with soap, atropine instillations, and moist heat several times daily, with a bandage all the time, will do a great deal of good.

The results of this treatment are very satisfactory.

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III. CAUTERIZATION.

The chemical, thermic, or electric cautery serves a very good purpose not only in diseases of the cornea, but also in diseases of the conjunctiva, the lids, the conducting lacrymal apparatus, etc. It is used almost exclusively to destroy tissue which is either exuberant, misplaced, infiltrated with infective substances, or invaded by pseudoplasms. In many cases our object is obtained with chemicals as well as with heat; in others both have advantages and disadvantages.

The *chemical caustics* are manifold, either strong acids or alkalies or salts. Among the latter, nitrate of silver in substance, either pure or mitigated with equal or double parts of nitrate of potassium, is the most serviceable, being used in corneal ulcers, inflamed or exuberant conjunctivæ, obliteration of the lacrymal sac, etc.

The *thermo-cautery* is a handy apparatus, and very popular.

The *galvano-cautery* is preferred by most physicians. It requires an electric—either immersion or storage—battery with a rheostat. Where electric light is in the house, it can, by means of a transformer, be used for galvano-cautery, and be connected with any conductor. The most convenient way is to connect the apparatus with the street current. A storage battery inserted into the conducting wire and a stationary rheostat secure a permanent current. I have used such an apparatus in my office daily these four years, and not once has it been out of order.

The *actual cautery* should be always at hand where there is no reliable galvano-cautery. It is very simple, consisting of a spirit-lamp and a thick platinum wire with a handle, and if this should be mislaid, a squint-hook will do as well. If the galvano-cautery is to be used, we should be careful not to have too strong a current, for this may melt the electrode, and, apart from that, it is mostly inadvisable to bring the burner to a white heat.

In using the galvano-cautery the eye should be well anæsthetized and held quiet, the burner held cold on the place to be cauterized; then by making the connection bring the burner to a deep-red heat, and quickly remove it. We can go from one place to the other, but the action should not be uninterruptedly continued longer than a few seconds, especially on the cornea, on account of the propagation of the heat to the aqueous and surrounding structures, in particular the lens.

Indications *of Cauterization, in Particular of Galvano-Causis.*

(1) The cautery is most extensively and very beneficially used in *infected corneal ulcers.* It was first tried for that purpose by Martinache, of San Francisco.[1] The publication of Gayet, in 1877,[2] drew general attention to

[1] Martinache, Ulcers of the Cornea treated by the Actual Cautery, Pacific Medical Journal, 1873, November, p. 294. De l'emploi du cautère actuel dans les maladies des yeux, et principalement dans les ulcères de la cornée, Annales d'oculistique, 1873, t. lxxx. p. 21.

[2] Gayet, Cautérisation ignée de la cornée, Gazette des Hôpitaux, 1877, No. 11.

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this mode of treatment, which has become very popular. Among the authors on this subject we may mention Dr. Nieden, who in 1884, at the twenty-fifth annual meeting of oculists at Heidelberg, and a year later in the *Archiv für Augenheilkunde*, Bd. xiv. S. 336, and Bd. xv. S. 405 (Engl. ed., *Archives of Ophthalmology*, vol. xiv. pp. 31 and 455), published a longer series of well-observed cases. The present writer is quite conversant with this mode of treatment, and recommends it highly for *pustular*, *fascicular*, *rodent*, *serpent*, and *deeper circumscribed purulent* inflammations of the cornea, without or with hypopyon. We use, according to the condition of the infiltration, a pointed or a circular burner. It is particularly to be mentioned that the infiltrated edges of the ulcer should be cauterized. In all severer forms the cauterization should be combined with perforation of the cornea, which perforation should be kept open by a probe as long as the bottom and edges of the ulcer show infiltration. More of this hereafter in the discussion of Saemisch's keratotomy.

(2) Galvano-causis is excellent in destroying the infiltration at the tip of *fascicular keratitis* (the so-called frenulum scrofulosum); further, according to Schuler, the *crescent that caps the pterygium*, if left after the removal of the fleshy conjunctival hypertrophy encroaching on the cornea; furthermore, the *remnants or roots of papillomas and other pseudoplasms* of the cornea, when the growth is abscised as cleanly as the operator thinks it permissible. For the former two a circular burner is suited, for the latter a flat, so-called surface burner. I have done this in several cases in which no relapse has occurred for from five to twelve years. The raw surface and its border are simply singed over with the thin, red-hot blade of the cautery quickly but thoroughly. It is remarkable how little reaction, if any, follows this procedure. In one case I cauterized in this way fully half the surface of the cornea; the recovery was by repair of almost perfectly transparent tissue, without any irritation, and there has been no relapse.

(3) In *keratoconus* my experience[1] coincides with that of A. Critchett and Tweedy,[2] who assert that no other treatment has given them such satisfactory results, a statement which is also endorsed by Panas.[3]

At first I used a pointed burner, and pierced the cornea freely. The recovery was tedious, but the result perfect and permanent. Then I made the cauterization very cautiously and pierced the cornea so slightly that the aqueous did not jet but oozed out. This took a longer time, and was followed in from six to eight weeks by the formation of a peculiar-looking yellowish cataract (the patient was fourteen years old). The wound healed well. I extracted the cataract later, and the eye has had good sight ever since, eight years. I have always considered this cataract as due to the overheating of the aqueous humor during the cauterization, for I had never seen such a peculiar-looking cataract apart from its developing in a young and healthy eye.

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[1] Knapp, Five Cases of Keratoconus treated with Galvano-Cautery, Archives of Ophthalmology, 1892, p. 540.

[2] Transactions of Ophthalmological Society of the United Kingdom, January 28, 1892.

[3] Traité des maladies des yeux, t. i. p. 293, 1894.

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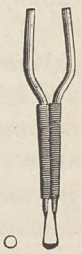
Later I used round burners, the tips of which were segments of the surface of a sphere, made for me in different sizes by E. B. Meyrowitz, New York. (See Fig. 19.) They can be used also for other affections. They are applied cold to the point of the cone, which is almost always below the centre of the cornea, and are then made red-hot, so as to produce a marked eschar and at the same time perforate the cornea. I have found that without perforation I had to repeat the cauterization once or twice, and the cornea did not flatten well. In this way, independently, I came to the conviction of Professor Tweedy, who considers the perforation essential to success.

Fig. 19.



The principle of curing keratoconus by cauterization was first expressed and put in practice by A. von Graefe. The cone has to be destroyed, and the subsequent cicatricial contraction will flatten the cornea. Graefe's procedure was to slice off a thin layer of the protrusion and cauterize the raw surface with the nitrate of silver stick. The operation was difficult of execution, and the results were uncertain in his as well as in other hands.

(4) In *infected wounds*, especially after *cataract extraction*, as well as in corneal ulcers infected by purulent conjunctivitis, galvano-causis has been widely used, and good results have been reported to the credit of this mode of treatment. I have practised it repeatedly, but cannot freely join in its praise. The good results in cataract extraction have been so uncertain that they were, to say the least, paralleled by those of the non-operative treatment, and when produced by purulent conjunctivitis the cauterization seemed to have no, at least no demonstrably, good effect on the recovery. They depended visibly on the course of the conjunctival disease, and the same cause, infection and reinfection from a diseased conjunctiva or lacrymal sac, seemed to have been at work in suppuration of the corneal section. I do not say that local treatment—*i.e.*, treatment directly applied to the infected wound—is useless; but I am satisfied that the treatment of the cause of the infection, the conjunctiva and lacrymal sac, is of paramount importance.

(5) *Galvano-causis* is used with advantage in *conjunctival disease*, especially for *trachoma*, where the granules are destroyed one by one: rather a tedious process. *Electrolysis* was used and recommended by G. Lindsay Johnson, of London, with good results, which have been confirmed by others who tried his method described in the *Archives of Ophthalmology*, vol. xix., 1890, p. 264. It consists in first scarifying the conjunctiva, then applying the electrode successively to all diseased parts.

Galvano-causis and probably electrolysis are useful in *exuberant granulation-tissue of the conjunctiva*, especially in *tuberculosis* (or *lupus*) of the conjunctiva, the lids, the tear-passages, and the nose. A few cases of this kind which I have treated with the galvano-cautery have been greatly benefited. I think that a permanent cure may be obtained by treating these cases thoroughly and persistently, as they are local affections, at least for a long time.

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(6) The galvano-cautery is used to *obliterate the lacrymal sac* by destroying its inner wall. I have tried it, too, for this purpose, but prefer in general chemical caustics and in particular cases extirpation. It seems that it is more difficult to destroy the whole extent of the sac with the cautery than with nitrate of silver and other chemical caustics.

(7) *Small staphylomas* of the cornea have been burned off with the galvano-cautery. I prefer abscission. Fuchs[1] recommends trephining and keratoplasty. Small and especially flat *prolapses of the iris*, as well as the *angular incarcerations* of iris after cataract extraction, and *cystoid scars* after extraction and iridectomy, may be advantageously treated with the galvano-cautery, especially when they show a beginning suppurative inflammation. Abscission and excision of these prolapses may very well be combined with galvano-causis. I have observed cases where destruction of the eye has been averted by early interference with these incarcerations.

IV. PARACENTESIS (PUNCTIO) OF THE CORNEA.

This little operation is made with a straight or bent lance, or with a narrow or triangular cataract-knife. Desmarres's paracentesis-needle, consisting in a small lance with a sudden thickening at the base to prevent the needle from penetrating too deeply into the anterior chamber, can be dispensed with.

Indications.

(1) *Evacuation of blood from the anterior chamber*, especially after traumatism and iridectomy for glaucoma. Blood in the anterior chamber is sometimes slow to absorb, and becomes dark and irritant even in eyes not predisposed to glaucoma. In such cases paracentesis is very beneficial.

(2) In accumulations *of pus in the anterior chamber* the paracentesis through the corneal ulcer or (less good) at another, somewhat lower, place is useful.

(3) In *iritis serosa*, especially when there is increased eyeball tension, it frequently does good service.

(4) In certain cases of *glaucoma* it is a useful palliative.

(5) In swelling of the lens after discission or an injury it at once relieves the symptoms, and if repeated hastens the absorption of cataract.

Technic.—The eye being cocainized, and the patient's head kept steady in a reclining position, the operator fixes the eyeball with one hand and thrusts the knife through the cornea with the other. The puncture should be from two to four millimetres long, two to three millimetres from the limbus, unless an ulcer requires another location. The aqueous may be evacuated while slowly withdrawing the knife and pressing on the posterior lip of the little wound, or the wound is held open with a probe.

There are no accidents during a carefully executed paracentesis. The object of the operation—the evacuation of blood and pus—may not always

[1] E. Fuchs, Über Keratoplastik, Wiener klinische Wochenschrift, 1894, xlv.

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be obtained, in which case a larger opening should be made, or some other treatment resorted to.

V. KERATOTOMY OF SAEMISCH.

We do not here consider incisions into the cornea as a step in other operations, for instance, in cataract extraction, removal of foreign bodies from the anterior chamber, and the like, but that kind which Professor Saemisch, of Bonn, in 1870, recommended for the treatment of a very dangerous kind of corneal ulcer,[1] which is characterized by its tendency to creep from its original, mostly centrally or slightly inferiorly situated, focus in different directions over the cornea, surrounded by an elevated border, infiltrated like its bottom, frequently accompanied by hypopyon, showing a great tendency to destroy the cornea, and not infrequently the eye. The **operation consists** in a *slitting of the cornea through and beyond the ulcer.* A narrow knife (Graefe's) is thrust one or two millimetres through the edge of the ulcer, passed through the anterior chamber, and through the border of the ulcer on the side opposite to its entrance. The back of the knife is turned to the iris and lens, and the knife carried carefully through the chamber and slowly out. Rarely is there hemorrhage into the anterior chamber, and the hypopyon is removed in most cases.

The operation is **indicated,** according to Saemisch, only in the severer cases, but in these yields better results than the other modes of treatment, —namely, the *mild treatment,* consisting in instillations of atropine, poultices, compressive bandages, boric acid instillations, slitting of the upper canaliculus, and frequent expression of the sac in simultaneous dacryocystitis, and the *caustic treatment,* consisting in cauterizing and sterilizing the ulcer and its border, as done by Fukala, de Wecker, Schiess-Gemuseus, etc. According to a recently published monograph on "The Treatment of the Ulcus Corneæ Serpens at Saemisch's Clinic," by Dr. Reiner Schmitz, assistant to the clinic,[2] there have been treated during the last seven years two hundred and sixty-one patients for ulcus corneæ serpens, 5.4% of the whole number of clinical patients. If the ulcer did not improve in from one to three days of hospital treatment, the keratotomy was made. The extent of the ulcer in which the operation was considered to be indicated varied from three to seven millimetres; at an average it was five millimetres. All operative treatment was omitted if the ulcer occupied almost the entire cornea. After the operation the eye was bandaged and the patient kept in bed. Atropine was instilled, the eye carefully cleansed, and if the ulcer, especially its raised edges, did not show a continuous improvement, the wound was reopened with a Weber canaliculus-knife every day or every few days until the ulcer cleaned itself and the inflammation subsided. The present writer has a good deal of experience in

[1] Saemisch, Das Ulcus Corneæ Serpens und seine Behandlung, Bonn, 1870.
[2] Bonn, 1897.

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these cases, and can only agree with the statement of Saemisch that, whereas the milder cases do well under the disinfectant methods, there is no better mode of treatment for the severer forms than the keratotomy, with repeated reopenings of the incision if it closes prematurely. The method is in harmony with the principles of modern surgery. Give vent to the infective material by the incision, keep the channel free by reopening the cut, and disinfect and drain the region of the wound: the latter is effected by the constant outflow of aqueous humor. The most important point in the treatment is the free division of the infiltrated border of the ulcer; in it the infective material is heaped up. If in a case the keratotomy was insufficient, it should, as in insufficient corneal section in cataract extraction, at once be increased by Stevens's scissors. Dr. R. Schmitz gives the results of the 126 cases treated at Saemisch's clinic by keratotomy during the last seven and a half years as 91 recoveries, 6% of total leucoma or anterior phthisis, and 3% of panophthalmitis, which is very satisfactory in so grave a disease as the one under consideration. It is not to be omitted, however, that keratotomy gives rise to more or less extensive anterior synechiæ, and occasionally to glaucoma.

VI. OPERATION FOR CORNEAL STAPHYLOMA.

A. *Partial Staphyloma.*—If the protrusion is small and recent, *simple incision* and occlusion of the eye may produce a permanent recovery. If it fails, an *iridectomy* should be made, by which, according to many earlier and recent writers (Beer, Rosas, Himly, A. von Graefe, Arlt), not only an improvement of sight but also the flattening of the protrusion may be obtained. Chelius, Sr.,[1] says, "Iridectomy converts partial staphyloma into a simple synechia, and prevents its further development."

De Wecker and others have removed partial staphyloma with the corneal trephine.

Abscission is the simplest method of partial staphyloma. Under local anæsthesia half the protrusion is detached from its base by an incision with a narrow knife, and the other half removed with forceps and scissors. Sutures need not be applied, but the eye should be kept closed and the patient remain in bed a few days.

Excision of the protrusion and its connections with the cornea, iris, and ciliary body, combined with galvano-causis of the wound, is the proper treatment for the partial staphylomas at the periphery of the cornea, as they so often occur after injuries and operations on the eye. Corneal fistulæ and lacunar or cystoid scars, with incarceration of the iris, form the indications of the above operation.[2] I have seen Professor Panas perform this apparently bold operation in a most thorough manner, and have since done it myself, with no appreciable reaction and with good permanent result.

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[1] Die Staphylome der Hornhaut, Heidelberg, 1847, S. 48.

[2] See Czermak, Über cystenartige Höhlen in Hornhautnarben, Archiv für Ophthalmologie, xxxvi. 2, S. 163.

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B. *Total Corneal Staphyloma.*—The following operations may be practised.

(1) *Küchler's operation* is a proper and efficient measure to prevent the development of staphyloma. When in sloughing of the cornea from purulent conjunctivitis or other causes the iris and cicatricial tissue began to protrude and sight was hopelessly lost, I saw A. von Graefe *run a cataract-knife through the protrusion from the temporal to the nasal border of the cornea, split the capsule, and let the lens out* according to Küchler. Under the same circumstances I have done this operation not infrequently, and can confidently recommend it. It does not favor but rather tends to prevent the development of panophthalmitis, abridges the course of the disease, and commonly leaves the eyeball unchanged in size, and the cornea replaced by a flat leucoma.

In the classical *amputation of staphyloma*, according to Beer, the lower half of the protrusion is detached with a cataract-knife, the upper seized with forceps and cut off with scissors. The lens should be let out after opening the anterior capsule. Both eyes are kept closed for two or three days, and the eye operated on for about a week. The patient has to be kept in bed as if he had been operated on for cataract. I have performed this operation often in previous years, and occasionally perform it still to show my pupils how readily and smoothly under ordinary aseptic precautions such an extensive wound of the eye heals. Beer says,[1] "Thus far I have made this operation one hundred and two times, and only three times the eye was lost by suppuration." Strings of connective tissue run over the wound, multiply, broaden, unite, and gradually close the wound with a white, unyielding scar.

(2) *Critchett's Operation.*—In order to protect better the interior of the eye, George Critchett[2] *closed the wound with sutures.* Before he incised the staphyloma he passed four or five long, curved needles provided with silk threads through the anterior part of the globe, introducing them through the upper part of the sclerotic and pushing them through the vitreous chamber behind the lens and through the lower part of the sclerotic, leaving them in the eye when their points had protruded four or five millimetres beyond the surface of the sclerotic. He then abscised the staphyloma, drew the needles out, and tied the threads so as to close the wound. The sutures were usually left in for from two to three weeks. At the time of the publication Critchett had performed the operation about thirty times; suppuration occurred in four cases.

(3) *Knapp's Operation.*—In order to avoid passing needles and threads through the interior of the eye, particularly through the ciliary body, the present writer closed the wound by external sutures, and published his method in 1868.[3] Before the staphyloma is abscised, a curved needle is

[1] Arlt, Operationslehre, Graefe-Saemisch's Handbuch, Bd. iii. S. 376.
[2] Royal London Ophthalmic Hospital Reports, vol. iv. p. 1, 1863.
[3] Archiv für Ophthalmologie, Bd. xiv. 1, S. 273.

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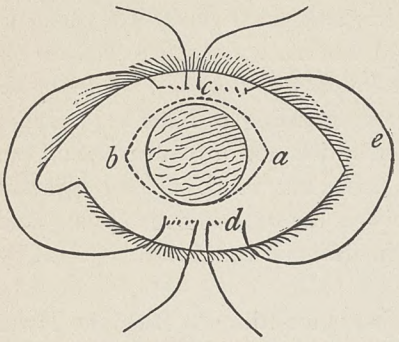
passed through the conjunctiva and the outer layers of the sclerotic from the upper end of the vertical meridian (Fig. 20, *b*) four millimetres behind the limbus, about four millimetres temporally, being drawn out and re-entered below the cornea (*d*), and passed through the conjunctiva and the superficial layer of the sclera in the same way as above the cornea, only in the inverse direction, coming out in the lower portion of the vertical meridian. The needle is then removed, but the thread left in position so that both its ends and a loop (*e*) between them are placed on the temple. Another thread is symmetrically applied to the nasal side of the episclera. The staphyloma is then abscised according to Beer (Fig. 20, *ab*). The lens is let out and the sutures are tied. The ends of the temporal thread are drawn nasally until the loop is converted into a straight line and the upper and lower lips of the temporal side of the wound meet. The same is then done with the nasal thread. It is seen that in this way the wound is closed by four vertical threads, each loop acting as a suture. When the sutures are tightly united the surface of the wound is no longer rectilinear, but more or less puckered, like the mouth of a purse. The wound closes perfectly. An ordinary bandage is applied, and the patient put to bed for a few days. The healing is generally undisturbed.

FIG. 20.



Abscission of staphyloma, with closure of the wound by external (sclero-conjunctival) sutures.

The wound can, of course, also be closed by four vertical sutures, as I said in my first publication (l. c., p. 27).

(4) *De Wecker's* operation[1] consists in covering the defect *with conjunctiva by a purse-string suture.* He detaches the conjunctiva round the staphyloma, and dissects it from the sclerotic towards and near the equator. Then he passes a thread through the free margin and ties it.

(5) *Brudenell Carter's* operation[2] covers the defect by detaching the tendons of the four straight muscles from the sclerotic and uniting by catgut sutures the external rectus with the internal and the superior with the inferior. The conjunctiva is drawn over them and united by four silk sutures in a vertical line. He was led to the device of this operation by the observation of a case of sympathetic ophthalmia after a Critchett operation.

(6) Panas[3] abscises the protrusion and closes the defect by four sutures passed through the sclerotic and conjunctiva from within outward.

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[1] De Wecker, Annales d'oculistique, lxix. 51, 1873, and Chirurgie oculaire, 1879, p. 188.

[2] Brudenell Carter, On an Improved Method of Abscission of the Anterior Portion of the Eyeball, Transactions of the Medico-Chirurgical Society, London, 1876, p. 193.

[3] Traité, i. 285, 1894.

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(7) Czermak[1] also advises to unite the scleral lips of the wound by from four to six vertical catgut sutures and cover them with conjunctiva by silk sutures. All these operations have for their object to prepare the eye in the best way for the prothesis of an artificial eye. There is no doubt that they furnish a better stump than all the other methods,—enucleation, evisceration, and implantation of a glass or metal ball. The latter can hardly as yet be considered to yield permanently good results. Usually foreign bodies of that size will work out sooner or later.

A noteworthy *accident* during the staphyloma operation is intra-ocular hemorrhage, to which eyes with secondary glaucoma, from which staphylomatous eyes frequently suffer, are predisposed. This intra-ocular hemorrhage throws the vitreous out and the retina and choroid into the wound. The eyeball, under excruciating pain, shrinks by subsequent *panophthalmitis* unless it is enucleated at once.

I should not omit to mention my belief that no staphyloma operation is absolutely devoid of the danger of awakening sympathetic ophthalmia, though I have never met with a case in my own practice. Two of the eyes on which I had performed a staphyloma operation in former years became tender and troublesome a number of years later, on account of which I thought it prudent to enucleate them. Panas[2] says that a staphyloma operation according to Critchett, if made before the insertion of the iris, is connected with no danger either immediate or subsequent. I think that the farther removed from the iris and ciliary body the wound and the sutures are held, the safer is the patient from irido-cyclitis in both eyes. For that reason I make the operation of staphyloma only in eyes that show no tendency towards irido-cyclitis, and among the operations I prefer those that secure the smoothest recovery and keep away from iris and ciliary body.

VII. KERATOPLASTY.

The cases of total leucoma with good perception of light have stimulated many a good surgeon to remove a portion of the scar and replace it by a transparent substance. As such, a disk of glass was used by Nussbaum, in 1856. It healed in and gave the person a modicum of vision for a short time, then it incrusted and was cast off. Similar experiments made before and after Nussbaum had the same fate, and are, it seems, definitively given up.

The transplantation of cornea from animal or man has had a more extensive trial from Reisinger, 1824, to 1888, since which latter date it has been very little heard of. These experiments have proved that transplanted disks of cornea can be inserted into healthy eyes of animals, take root, and remain perfectly transparent for a time, of which, in experiments of Dr. Silex on dogs, I have convinced myself. How long they remain trans-

[1] Die augenärztlichen Operationen, 1896, S. 613.

[2] Traité des maladies des yeux, t. i. p. 268.

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parent I know not. In man the operations of Professor von Hippel have been the most successful, or rather have been less unsuccessful than the others. At the Heidelberg Ophthalmological Society in 1887 he showed a patient on whom he had made a keratoplasty about a year previously. The implanted piece was still tolerably transparent, and the patient had useful sight. How long he enjoyed it I do not know. The operation is not indicated in total leucoma, nor in partial leucoma when the periphery of the cornea is sufficiently clear to admit of an artificial pupil, nor is it indicated if the central opacity occupies the whole thickness of the corneal substance. This limits the applicability of the operation to very few cases, and I am not informed that permanently available sight has been obtained in any. The experiments and operations of von Hippel and many others deserve all praise as to ingenuity and perseverance, though the practical results have been meagre. If even a temporary success, useful vision for some years, could be expected, the operation would go out of the laboratory into the practice of ophthalmology.

The **operation is done** in the following way. With a corneal trephine, turned either with the hand (Bowman) or with a spring (von Hippel), a piece not exceeding five millimetres in diameter is punched out of the whole thickness of the cornea, and a corresponding piece from another cornea is inserted into the defect. If only the anterior layers are to be removed, a trephine incision is made through them, and the disk seized with delicate forceps and exsected with a bent lance-shaped knife. A similar disk from another eye is then implanted. No suturing. The eyes are kept closed by bandages for several days. For details the reader is referred to an extensive presentation of this subject in Czermak's book, "The Ophthalmic Operations," Vienna, 1896, p. 614, etc.

VIII. TRANSPLANTATION OF CONJUNCTIVA TO THE CORNEA.

Schöler, in the annual report of his clinic for 1877,[1] recommends the transplantation of pedunculated conjunctival flaps for *perforating and non-perforating ulcers, gaping wounds* (with or without prolapse of the iris), *fistulas, cystoid scars,* and *staphylomas of the cornea.*

Kuhnt[2] has transplanted unpedunculated flaps of conjunctiva on *ulcers of the cornea* which he had carefully cleansed before. He found that they cured the ulcers rapidly and prevented prolapse of the iris as well as opacities of the cornea. He also[3] has covered peripheric ulcers of the cornea with flaps from the adjacent conjunctiva. The flaps, which have a pedicle and are larger than the ulcer, are pressed on the ulcer with a spatula. The

[1] See also Schöler, Über das Pterygium von Conjunctivallappen, Berliner klinische Wochenschrift, xlvi., 1877.

[2] Bericht über die siebzehnte Versammlung der ophthalmologischen Gesellschaft, Heidelberg, 1885, S. 219.

[3] Kuhnt, Vorschlag eines neuen Weges zur Behandlung gewisser Formen von Cornealgeschwüren, Wiesbaden, 1884.

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eye is kept closed with a bandage for three days. Both Schöler and Kuhnt assert that the pain ceases very soon after the transplantation.

Da Gama Pinto[1] proposes to close the wound, after abscission of prolapsed iris, with a conjunctival flap without a pedicle. The flap is pressed with a blunt probe into the wound, its raw surface touching the wound.

With all these operations I have no personal experience. Knowing that corneal wounds (even with prolapse of the iris) as well as scleral wounds usually heal smoothly when the patient's eyes are kept quiet, I have been in the habit of treating them expectantly in the majority of cases. An aseptic corneal ulcer, I think, needs no conjunctival flap to cover it, and whether it is prudent to cover an infected ulcer, however carefully sterilized, has to my mind to be proved by further clinical observation, although L. Weiss[2] asserts that such misgivings have proved unfounded.

IX. TATTOOING OF THE CORNEA.

The origin of this procedure to conceal corneal opacities is old.[3] Galen treated them with tannate of iron. In modern times it was introduced chiefly by de Wecker.[4] Alt[5] and Hirschberg[6] have found that the pigment accumulates only in the anterior layers of the cornea, *i.e.*, in the deeper epithelial stratum, but chiefly in the superficial layers of the stroma. The grains are located in the interfibrillar spaces, in the fixed and movable cells, and in the walls and the endothelium of the vessels. Pigmented thrombi have been found in such vessels.

The operation is suitable only in old leucomas of unirritable eyes.

Technic.—The best substance is India ink, which should first be tested on eyes of rabbits for its harmlessness. It should be sterilized and rubbed up to an oily consistence in a sterilized mortar. The eye must be free from irritation and the conjunctiva aseptic. At first the ink was introduced obliquely into the cornea by *a grooved needle*, many times at the same sitting. To produce a satisfactory effect the pigment was deposited in short lines. The operation had to be repeated several times at an interval of one or several weeks. At present a pencil of from *four to eight round needles* is used. The cornea is well sterilized, and the eye steadied with a forceps the prongs of which are covered with hard rubber or bone to prevent tearing, which would stain the sclerotic. The needles are thrust obliquely into the cornea a great many times, so as to destroy the epithelium completely

[1] On the Treatment of Prolapse of the Iris in Corneal Ulcers, Klinische Monatsblätter für Augenheilkunde, Bd. xxv., 1887, S. 1.

[2] L. Weiss, Archiv für Augenheilkunde, Bd. xxxiii. S. 314.

[3] See a complete bibliography by Hirschberg in Centralblatt für Augenheilkunde, 1891, S. 247.

[4] Tatouage de la cornée, Union Médicale, Mars, 1870; Chirurgie oculaire, p. 181.

[5] Alt, On the Microscopic Changes found in a Tattooed Cornea, American Journal of Ophthalmology, vol. i., 1884, p. 8.

[6] Hirschberg, Zur Hornhautfärbung, Archiv fur Augenheilkunde, Bd. xxviii., 1, 1884, S. 269.

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and make a great number of small canals in the corneal substance for the reception of the ink, which is now freely rubbed in with the finger. After this the cornea is irrigated with a warm sterilized solution of chloride of sodium (7 to 1000). If the coloration is sufficient, the operation is completed; if not, the needling, rubbing, and irrigation are repeated until the coloration leaves nothing to be desired. In this way a permanently good result may be obtained in one sitting.

The operation is usually followed by some circumcorneal injection, but this and all irritation soon disappear. Not infrequently the coloration diminishes in the course of years, even when done by experts. Inflammation and even symptoms of sympathetic inflammation are mentioned as sequels of this cosmetic operation. (Panas.[1])

§ V. OPERATIONS ON THE CONJUNCTIVA.

1. MINOR OPERATIONS.

A number of small operative interferences, such as scarification in severe blennorrhœal conjunctivitis; puncture or incision in unusually large subconjunctival hemorrhage; excision of small tumors, such as pingueculæ, vascular or pigmented nævi, lens dislocated under the conjunctiva, and the like; suturing of the small defects resulting from such excisions; the instrumental, chemical, or thermic destruction of conjunctival granulations and excrescences, for instance, the granulomas after spontaneous or artificial opening of Meibomian hordeola and chalazia, or after operation on the tendon or sclerotic or from tuberculosis, amyloid degeneration, or other conditions,—all require no special description, as the rules of general surgery are sufficient to determine how to perform these little operations without interfering with the shape or function of any part of the eye. The ophthalmic more than the general surgeon will, in such minor operations, always fully realize that even small injuries or cicatricial connections may in an organ so small and delicate as the eye produce inordinate disturbances, such as epiphora, diplopia, etc.

II. REMOVAL OF SUBCONJUNCTIVAL TUMORS.

(1) *Serous and echinococcus cysts* are sometimes met with in the subconjunctival tissue. Their extirpation should be carefully done, so as to remove the containing bag, if possible, unbroken. It is best to raise a small fold of conjunctiva with delicate forceps, snip it, and lay the tumor bare. The shelling out can be done with a scalpel or a delicate, slightly curved hand-chisel, the edges of which are rounded and, like the adjacent portions of both sides, sharpened. (See Fig. 21.) This chisel is particularly useful in freeing the deeper parts from the neighboring tissue and prying the tumor out. The wound, which nearly always heals by first intention, may be stitched.

(2) *Subconjunctival lipoma* is not very rarely met with. Besides fat the

[1] Traité des maladies des yeux, t. i. p. 275.

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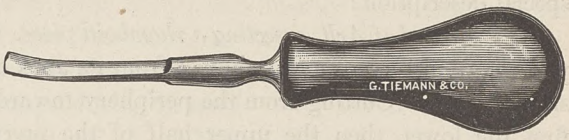
growth contains a small amount of fibrous tissue and blood-vessels. Its favorite seat is the upper-outer corner of the conjunctiva, where it forms a flat swelling, sometimes visible through the lids. During its removal the patient should look down, and the upper lid be raised and drawn off from the eye by the finger of an assistant. Sometimes these tumors become the size of a large French bean and reach below the external canthal ligament. Furthermore, as most of them have no well-defined capsule, and their hardened fibrous surface is firmly connected with the conjunctiva, it is not easy to shell them out. The best way to remove them is to dissect the conjunctiva from the tumor, which on the front part is sometimes slow work; in the depth the growth imperceptibly loses itself in the orbital fat. As soon as only fat comes out on pulling the tumor forward, the operation may be terminated by cutting off the part connected with the growth, and pushing the remainder back into the orbit. The wound is then cleansed and closed by several fine sutures. A bandage should be applied for several days.

FIG. 21.



Hand-chisel.

Sequels.—I have not seen any accidents during or immediately after the operation, but I have seen two unpleasant sequels,—*suppuration* and *diplopia*. The former was of my own doing, the second was not. The operation performed on my patient, a boy of about six years, was very laborious. It was over twenty-five years ago, when antisepsis was not in use. The tumor was very large and far forward towards the cornea. It came out well, and I had no apprehension. On the third day suppuration in the wound was manifest and a corneal ulcer formed. I took him to the hospital, kept him in bed, made cold applications as long as the eye was red, swollen, and painful, cleansed the eye carefully, and used a mild astringent wash. The inflammation subsided and the corneal ulcer healed, leaving a superficial opacity in the outer third. The boy is now a grown man. Recovery complete. Sight and binocular fixation normal.

The second case was seen in consultation. The region of the wound was swollen, but there was no suppuration. The adduction was diminished. The inflammation disappeared in a few weeks. The adduction remained diminished, and there was diplopia. The eyeball was connected with the outer orbital wall by an unyielding swollen scar.

I mention these cases to show how important it is to perform even the smallest operations neatly and with full appreciation of the delicacy of the organ into which we put our instruments.

III. PTERYGIUM.

A great deal has been written on the nature and formation of pterygium, and the different views and hypotheses have not been without effect on the

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methods of operating for this anomaly. As long as pterygium was considered a tumor it was simply excised. Acrel[1] circumcised its head and extirpated it totally, Richter and Scarpa only its extremity. Coccius[2] covered the defect by conjunctival sutures. The following methods seem to deserve special description:

(1) *Method of Arlt, exsecting a rhomboid piece.* The pterygium is grasped with fixation forceps and removed from its apex on the cornea with a lance-shaped knife. Cutting from the periphery towards the centre of the cornea, first the lower, then the upper half of the pterygium is cleanly detached from the cornea. After that a triangular piece of the bulbar part of the pterygium is exsected, the base at the corneal margin, the apex near the caruncle. The defect is covered by a conjunctival suture.

If the pterygium is small, Arlt makes only two convergent incisions into the bulbar portion, without removing the dissected part, which remains behind the suture and gradually disappears by atrophy. A. Pagenstecher operated in the same manner.

(2) *Szokalski's method, ligation of the epibulbar part.* A thread, armed with a needle on each end, is passed first near the cornea, then, with the other needle, three or four millimetres farther back, so between pterygium and sclera that the thread remains double at each end, single in the loop. Both needles are cut. One single thread is tied over the pterygium near the cornea, the other near the caruncle, so as to constrict the pterygium. Then the loop is tied so as to constrict the base. The portion between the threads is necrosed and the portion on the cornea wastes away, leaving an opacity.

(3) *Desmarres's method, transplantation into the lower fornix of the conjunctiva.* The pterygium is removed with a cataract or lance-shaped knife from its base to its apex, a curvilinear incision is made with curved strabismus-scissors into the conjunctiva of the lower fornix, and the apex of the pterygium is stitched into the angle of the incision. The eye is kept bandaged for several days. The defect in sclera and cornea soon covers itself with epithelium, and the transplanted mass of the pterygium shrinks by the pressure of the lower lid.

(4) *Knapp's*[3] *method, double transplantation and covering the defect by conjunctival flaps.* The present writer, having tried Desmarres's method and found it satisfactory in small pterygia, used it for larger pterygia with two modifications.

Technic.—The epibulbar portion of the pterygium is seized and slightly raised with a pair of fixing forceps; the pterygium is then detached with a narrow cataract-knife from the sclera to its apex, boldly removing all epicorneal tissue, even cutting through the superficial corneal layers, coming

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[1] Richter's Chirurgie, Göttingen, 1771, S. 97. Quoted after Panas.
[2] Rüte's Lehrbuch der Augenheilkunde, 1854, Bd. ii. S. 191.
[3] Archiv für Ophthalmologie, Bd. xiv., 1. Abtheilung, 1868, S. 267.

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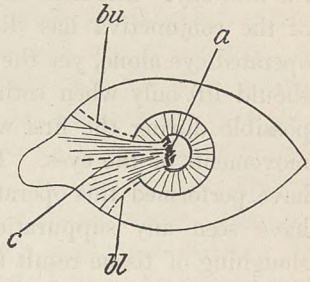
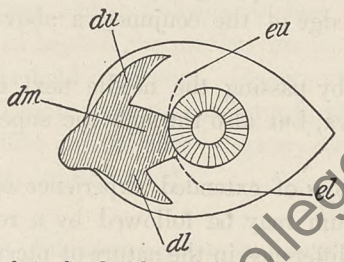
out beyond the gray cap of the triangular band (Fig. 22, *a*) so that nothing of it is left. The more thoroughly the epibulbar portion is removed the less opaque is the subsequent scar and the more surely is a relapse prevented. The bulbar portion is circumcised with curved scissors in a line running along its lower border (Fig. 22, *bl*), and another one along its upper (*bu*) towards its insertion into the semilunar fold, the former prolonged into the lower, the latter into the upper fornix, producing in each an angular gap (Fig. 23, *dl* and *du*). The pterygium is detached from the sclerotic, and the gray seam at its head cut off. Then it is split with a pair of straight scissors into symmetrical halves by a horizontal incision (Fig. 22, *c*) through its midst. The tip of the lower half is stitched into the apex of the triangular defect in the lower conjunctival fornix (Fig. 24, *sl*), and the upper half transplanted in like manner upward (Fig. 24, *su*). The defect in the bulbar conjunctiva is covered as follows. An incision is made through the conjunctiva upward, and another downward (Fig. 23, *eu*, *el*), beginning near the cornea and curving the one up, the other down towards the vertical meridian, not close to the corneal margin, but removing from it the more they advance. The conjunctiva lying between these incisions and the transplanted halves of the pterygium is undermined, forming two flaps (Fig. 24, *fl*, *fu*), which are stitched together at their ends by two sutures, the one at the corneal (Fig. 24, *m*), the other at the caruncular corner (Fig. 24, *n*). The latter suture should com-

FIG. 22.



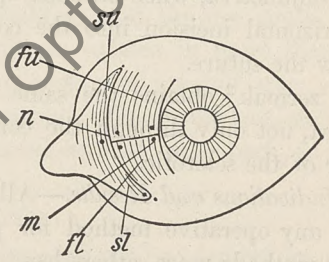
Pterygium: lines indicating detachment and splitting.

FIG. 23.



Defects after detachment of the pterygium.

FIG. 24.



Pterygium operation after transplantation of the flaps and covering of the defect.

prise the tissue forming the apex of the angle between the two halves of the pterygium, as indicated by a third point in Fig. 24. This attachment of the flaps to the base of the pterygium not only covers the angle between the transplanted halves of the pterygium, but prevents the united conjunctival flaps from overlapping the adjacent part of the cornea.

In this way the whole pterygium is removed. No portion of it, except the infiltrated crescent of corneal substance at its head, is cut away. The

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double transplantation obviates the deformity which would result from heaping a mass of hyperplastic tissue in the inner canthus, by dividing it symmetrically, transplanting and hiding one half under the upper, the other under the lower lid, where they will gradually shrink.

The operation is laborious. The conjunctiva in the region of the wound is somewhat swollen. Both eyes are covered with a roller bandage the first day. The next day, when the dressing is changed, all the swelling of the conjunctiva has disappeared. The bandage is now put over the operated eye alone, yet the other is covered with a patch which the patient should lift only when eating. He is advised to use his eyes as little as possible during the first week, in order not to disturb the sutures by the movements of the eyes. The sutures are taken out in five or six days. I have performed this operation often, for more than thirty years, and never have seen any suppuration or other inflammation nor hypertrophy or sloughing of tissue result from it.

(5) *Galezowski's method, stitching the loosened pterygium under the caruncle.* Galezowski detaches the pterygium from the cornea, passes a suture through its apex, doubles the pterygium up, and stitches the head to its base under the caruncle. The defect is covered by conjunctival sutures. The healing is smooth. The pterygium gradually shrivels.

B. Bettman, of Chicago, has of late published in different places the same operation, in which I see nothing new but the name he gives it,—viz., *subvolution.*

Schuleck,[1] of Buda-Pesth, recently published an article in which he recommends detaching the pterygium and letting it lie in the inner canthus after having closed the episcleral defect with sutures, exactly as Arlt and A. Pagenstecher did years ago. (See p. 836.) In order to prevent the conjunctiva, when stitched up, from overlapping the cornea, he makes a horizontal incision into the corneal edge of the conjunctiva above and below the suture.

Czermak[2] obtains the same effect by passing the needle next to the cornea, not only through the conjunctiva, but also through the superficial layer of the sclerotic.

Indications and Results.—All operators of extended experience confess that any operative method for pterygium may be followed by a relapse, some methods more, others less. The difference in the nature of pterygium may be the reason. It is well known that in very many cases the pterygium encroaches but little upon the cornea and then becomes stationary. Fuchs, of Vienna, who has made very thorough investigations on the pathology of pinguecula[3] and pterygium,[4] considers the two as analogous

[1] Zur Operation des Pterygium, Orvosi Hétilap "Szemészet," 1894, Nr. 4.
[2] Augenärztlichen Operationen, 1896, S. 661.
[3] Graefe's Archiv für Ophthalmologie, 1891, Bd. xxxvii., 3, S. 143.
[4] Ibidem, 1892, Bd. xxxviii., 2, S. 1.

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formations, depending chiefly on processes of involution, the connective tissue and elastic fibres of the subconjunctival and episcleral tissue, even the superficial layers of the sclerotic, having undergone hyaline degeneration. The epithelial strata of the conjunctiva do not participate in the hyperplasia and subsequent degeneration. He says that the true pterygium originates in a pinguecula. Fuchs is a man who knows what he says, but this statement is too sweeping to be accepted without reserve. Clinically these two conditions differ from each other as much as malignant tumors differ from benign. Pinguecula is almost always non-progressive; no surgeon would touch it unless to gratify the vanity of a frivolous person; whereas pterygium is progressive, if not in the majority, at least in a large minority of the cases. These minority cases may be akin to pinguecula, and need not be operated on, as long as they do not interfere with sight. They may constitute the bulk of those which, when removed, do not relapse. Yet pterygium is not a malignant disease. Its growth is slow, even in the worst cases, though I have to concur with Arlt when he says that "authors who pretend that pterygium rarely advances as far as the centre of the cornea, never beyond it, must have seen little."

As to the result of the pterygium operation, I can say that I do not remember, from my own practice, a single case in which the pterygium recurred to the same extent as before the operation. In the worst relapses the pterygium extended a little over the margin of the cornea and then remained stationary. The majority of the cases showed slight thickening in the old track, with some stretching of the semilunar fold, without discomfort or notable disfigurement. I have seen in my consultation-room a few cases with unusually bad relapses, cases that had been operated on both in America and in Europe. The hard fleshy mass was very disfiguring, and so tightly stretched and unyielding that the eye could not be moved beyond the median plane, and diplopia existed in more than half of the field of vision. It gave me the impression of a *keloid scar*. I advised some patients against a further operation. The result was, they took the first transatlantic steamer, were operated on, and came back, to put it mildly, unimproved.

CONCLUSIONS.

(1) Stationary pterygia, like pingueculæ, need not be operated on, except for cosmetic purposes. I consider this latter indication legitimate, because the operation under these conditions is harmless.

(2) A pterygium should not be operated on as long as there is an infectious condition in the conjunctiva or the lacrymal sac, or any irritation whatsoever present.

(3) A relapse of a pterygium should not be subjected to a new operation for a long time, say for one or several years, when all irritation, congestion included, will have long subsided.

(4) Pterygia that have relapsed after one or several operations and

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have the aspect of a keloid scar should not be meddled with. The patient should be made acquainted with the reasons of our apprehension, and also with the fact that pterygia when left alone, and when the eyes receive mild antiseptic treatment and good care, are likely to lose their angry looks, shrivel to a certain degree, and then remain stationary.[1]

(5) When we operate the eye should be free from irritation, and we should select the method according to the case, for small pterygia the simple detachment, with or without excision, or stitching the apex back. I have seen good results from all of these methods, but prefer for small pterygia the single transplantation, after Desmarres, for larger ones the double transplantation as described above.

(6) In all cases the pterygium should be totally removed from the cornea and sclera, especially the infiltrated crescent at its head. If this has not been done thoroughly from the start, it should be done afterwards, with a knife or the cautery. (See page 824.)[2]

(7) In all cases the episcleral defect should be covered with conjunctiva, both eyes bandaged several days, and the patient stay in his room, not use his eyes, and have company as little as possible. This restriction in so small and innocent an operation may appear uncalled for, but every movement of the eyes pulls at the sutures and diminishes the chances of a rapid and firm healing by first intention. Rest puts the patient in the very best conditions of having a perfectly satisfactory result with no relapse.

IV. DERMOID TUMORS.

Dermoid tumors, which mostly are situated on the temporal side of the corneo-scleral border, are congenital. Growing very slowly, they rarely attain greater dimensions than from two to three millimetres in elevation and one centimetre in diameter. They can and should be removed as soon as the child has passed its first year. They grow into the depth, and if removed incompletely leave a white scar. The most convenient instrument is a Graefe cataract-knife, with which they are exsected thoroughly. The incision may begin at the border; the portion first loosened, held with forceps, serves also to steady the eyeball, and with sawing motions of long excursion the rest of the growth is thoroughly excised. If we have cut too superficially, we must cut more until all white tissue has disappeared. In case this is impracticable, we may burn the white remnants with the galvano-cautery. There is no need to cover the defect with conjunctiva. The healing is smooth, and there is no relapse to be feared.

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[1] Dr. Darier recommends for that purpose massage with lanolinum hydrargyricum. See Recueil d'Ophtalmologie, 1889, p. 229, and Panas, Traité des maladies des yeux, t. ii. p. 263.

[2] Dr. A. E. Prince, of Springfield, Ill., recommends making an incision under the episcleral portion of the pterygium, passing a squint-hook through it, and tearing off the corneal portion of the pterygium with it. He alleges that in this way he removes the tip of the pterygium more easily and more thoroughly than with other methods.

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V. OSTEOFIBROMA.

A few cases of osteofibroma in or under the conjunctiva are on record.[1] They were removed. Healing smooth. As they all were small, from three to five millimetres in diameter, and situated near the outer commissure, they may be considered akin to the dermoid growths.

VI. TUMORS OF THE CARUNCLE.

The caruncle is the seat of various tumors,—fibromas, papillomas, dermoids, adenomas, cystadenomas, sarcomas, and carcinomas. Extirpation, which, owing to the exposed situation of the caruncle, is easy, should be done without delay. The character of these tumors as to malignity is sometimes not easy to ascertain. Granulomas, papillomas, adenomas, and sarcomas may be difficult to distinguish, both clinically and microscopically, and even if there have been relapses after previous operations this is not absolute proof of their malignity. The relapsing tumors should not be left to grow, but be removed more thoroughly, in particular cut out freely within the surrounding healthy tissue.

A man about fifty years of age came to me fifteen years ago with a soft, highly vascular tumor the size of a large cherry, with slightly nodular surface, occupying the region of the caruncle. He said it began as a small flesh-colored elevation on the caruncle, increased steadily, and was cut off when it had reached the size of a cherry-stone. It soon returned, and was removed when it had reached the size of a small cherry. All the fleshy part in the inner corner of the eye had been cut off. The tumors and the recovery were free from pain and inflammation. The last time the growth had returned sooner and increased faster than the first. The tumor was well circumscribed, not diffusely extending into the neighboring tissue, and was not exulcerated, all of which determined me to advise a third operation. On his ready consent I removed the growth and the surrounding apparently healthy tissue down to the lacrymal sac and the sclerotic over and before the tendon of the internal rectus. These parts appeared normal. I covered the denuded sclerotic with conjunctival flaps taken from above and below. The recovery was smooth. The tumor consisted of small, round, and fusiform cells supported by a scant connective-tissue matrix. Microscopically it was impossible to ascertain whether it was a granuloma or a sarcoma. The clinical observation decided in favor of the former. The man, of whom I received news for eight years, had no other relapse. The other eye was blind.

VII. OPERATIONS FOR SYMBLEPHARON.

The operations for symblepharon have received a good deal of attention from the profession. Their success depends chiefly on the quantity of conjunctival tissue that is preserved. We may distinguish two groups:

(*a*) *Symblepharon with limited or no Destruction of Conjunctiva.*—These forms are met with after injuries, burns, and the destructive conjunctival and corneal inflammations, especially diphtheria and blennorrhœa. To *prevent*

[1] (1) Von Graefe, Klinische Monatsblätter für Augenheilkunde, 1863, S. 23; (2) De Wecker, Traité des maladies des yeux, 1878, t. i. p. 427; (3) Critchett, Transactions of the Ophthalmological Society of the United Kingdom, May 11, 1882; (4) E. Loring, Transactions of the American Ophthalmological Society, July 26, 1882; (5) Vignes, Bulletins et Mémoires de la Société française d'Ophtalmologie, 1889.

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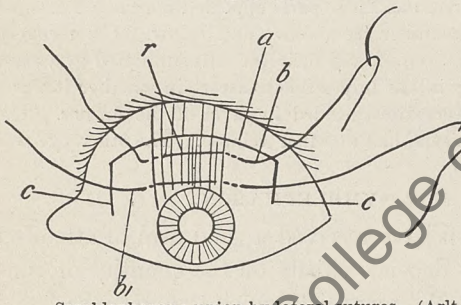
the union between lid and eyeball it may serve a good purpose to separate the raw surfaces several times each day in case the fornix portion is not ulcerous or mechanically denuded. As soon as the raw surface is covered with epithelium on one side there is no longer any danger of reuniting. If by this treatment the adhesions cannot be entirely prevented, their extent may be less than by merely expectant treatment. During the period of the daily separations the raw surfaces will, according to their tendency of covering themselves from the periphery, become constantly smaller and the symblepharon will be less broad. In blennorrhœal conjunctivitis with ulcers of the cornea along the upper or lower border, I would not touch the surface of the lids with a strong solution of nitrate of silver (one per cent. and more) or any other caustic that is apt to destroy, even if only temporarily, the epithelial protection of the lid. We must not forget, however, that during the whole disease our chief object is to limit the corneal destruction.

If the symblepharon is firmly established we leave it alone for some time, and deal with it afterwards, when all irritation is past. In case the fornix portion is free, which we ascertain by passing a probe underneath, *simple splitting of the bridge* is mostly sufficient. In case the symblepharon is somewhat extensive, we may cover the defect in the bulbar conjunctiva by stitching conjunctiva, loosened from the sides, over it.

More difficult to cure are the cases in which *the symblepharon extends from the cornea back to the fornix.* We have then to ascertain how broad the union in the fornix is. The operations in such cases are:

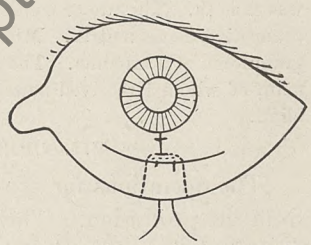
(1) Arlt's first method:[1] *Union by lateral sutures.* The adhesion is detached, and the cornea, if involved, cleared of all deposits. The raw surface (Fig. 25, *r*) is covered by drawing the conjunctiva from both sides over

FIG. 25.



Symblepharon: union by lateral sutures. (Arlt.)

FIG. 26.



Symblepharon: corneal portion detached and stitched into the fornix with threads passed through the lid. (Arlt.)

the defect with two sutures, one (*b*) near the fornix (*a*), the other (*b'*) two or three millimetres behind the cornea. If the conjunctiva is too much stretched, one or two *relaxing incisions* (*c*, *c*) are made in the conjunctiva.

[1] Operationslehre, in Graefe-Saemisch's Handbuch der gesammten Augenheilkunde, Bd. iii., 1874, S. 438.

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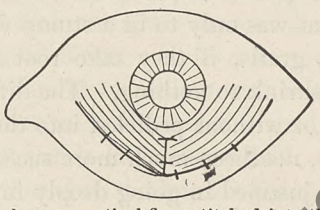
Arlt had done this operation in seven cases when he described it (*loc. cit.*), "in two on the upper side, in all of them without accident and with essential improvement, in small adhesions with complete recovery."

(2) Arlt's second[1] method: *Corneal portion transplanted into lower fornix by a loop-suture through the lid.* When the cornea was encroached upon in the manner of a pterygium, he detached the deposit from the apex down to the fornix and stitched the head with a doubly armed thread into the fornix (Fig. 26), passed the needles through the lid, and tied the ends of the thread on the outer side over a small cylinder of india-rubber. He thus used the flap for covering the tarsal surface. The bulbar denuded surface also was covered with lateral sutures (Fig. 26).

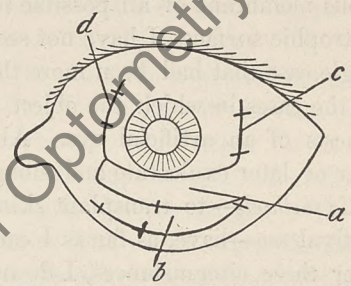
(3) Knapp's method: *Covering the defects with vertical, stretched flaps, stitched into the lower fornix.* I have done this and similar operations a number of times with different modifications which naturally suggested themselves according to the breadth of the defect and the quality of the preserved conjunctiva. I have never used relaxing incisions, which does not mean, however, that I condemn them. When the defects on the sclerotic were large, I made two horizontal conjunctival flaps, and prevented them from overlapping the cornea by uniting their lower border with the submucous tissue of the fornix, or passed the sutures through the lid and tied them outside, according to Arlt. Sometimes, when the defect was very large and but little conjunctiva left to draw upon, I carried the incisions (Fig. 27) upward and slightly outward,[2] united the vertical edges

FIG. 27.



Symblepharon: vertical flaps stitched into the fornix. (Knapp.)

FIG. 28.



Symblepharon: transplantation of vertical flaps (*a*, *b*) to cover the defects on the globe (*a*), and on the lid (*d*). (Teale.)

of both flaps by sutures under the cornea, and stitched the lower borders into the conjunctival fornix.

A. Alt,[3] in a case where the symblepharon encroached upon the cornea, was very dense, and broadened towards the fornix, made also two upward

[1] Operationslehre, in Graefe-Saemisch's Handbuch der gesammten Augenheilkunde, Bd. iii. S. 439.

[2] Knapp, Operation eines fast totalen Symblepharons des unteren Lides, Graefe's Archiv für Ophthalmologie, Bd. xiv., 1, 1868, S. 270.

[3] A. Alt, An improved method of operation in certain cases of symblepharon, Archives of Ophthalmology, 1880, vol. ix. p. 293; Archiv für Augenheilkunde, Bd. x. S. 322.

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flaps, first concentrically around the cornea, then diverging above. He fastened the lower surface of the flaps by two loop-sutures, which he carried through the lid and united outside over glass beads.

(4) Teale's symblepharon operations are ingenious. In his *first method*[1] he covered, in a case of downward symblepharon, the defect on the eyeball by an upward flap taken from the temporal side (Fig. 28, *a*), and the defect on the lid by an upward flap taken from the nasal side of the bulbar conjunctiva (Fig. 28, *b*). Both flaps were twisted 90 degrees, and the bare surfaces left by their transplantation were covered by drawing the adjacent conjunctiva over them with horizontal sutures (Fig. 28, *c*, *d*).

(5) In his *second method*,[2] Teale covered the defect, in a case of downward symblepharon, by *a curved strip of conjunctiva taken from the lateral and upper bulbar parts, its ends remaining attached to the sides.* The strip was dissected from its submucous stratum and shifted unbroken down over the cornea, where its upper margin was stitched to the remnant of the pseudo-pterygium at the corneal border, and the lower margin was stitched into the fornix as deeply as possible.

The consideration of these methods will show in what way symblepharon may be dealt with when only a portion of conjunctiva is destroyed.

A vastly more difficult problem is before us when

(*b*) *The conjunctiva is more or less atrophied.* More than twenty years ago[3] Arlt said that cases of total atrophy are for the surgeon a *noli me tangere.* In spite of the numerous attempts which during the last twenty-five years have been made to remedy this unfortunate condition by grafting skin or mucous membrane of all possible localities and in all possible manners on the atrophic surfaces, I have not seen, either in New York or in my travels, a single case that had been more than a temporary improvement, including even the cases in which the object aimed at was only to fit a stump for the prothesis of an artificial eye. All these grafts, if they take root at all, sooner or later exulcerate and slough or shrink to nothing. The different *plastic methods*—to transplant skin, with or without pedicles, into the conjunctival sac—have, as far as I can judge, not been much more successful. Under these circumstances, I do not feel justified in going deeply into details in describing the different, perfectly legitimate, trials; yet, as science is ever progressive, I shall endeavor to give a comprehensive account of what has been done in this direction.

The older methods of securing free space by putting between the *wounded surfaces a foreign body*—*e.g.*, plates of metal—have been abandoned as useless.

The method of Himly, to make a canal through the cicatricial tissue uniting eyeball and lid, by inserting a *lead nail through the fornix* and keeping it there for months, has proved a failure.

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[1] Teale, Royal London Ophthalmic Hospital Reports, vol. iii. p. 253.

[2] Teale, London Congress (1873) Report, p. 159.

[3] Graefe-Saemisch's Handbuch der gesammten Augenheilkunde.

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The grafting of *small pieces* of skin or mucous membrane, according to Riverdin, successful on skin wounds, had practically to be given up when tried on the conjunctiva. The unavoidable movements of the eye almost always prevented them from taking root.

The grafting of *larger pieces* of mucous membrane, first done by Stellwag, of Vienna (1873), and Wolfe, of Glasgow (1872), "does," according to a statement of Stellwag in a later publication,[1] "not yet in any way answer the requirements we could reasonably expect." As to the technic and the value of grafting *mucous* membranes on the conjunctiva, I may refer to a paper by Wölfler in *Langenbeck's Archiv*, xxxvii., No. 4.

Transplantation of skin-flaps has had, it seems, a modicum of success. It has been tried by different oculists.

Post[2] took the flap from the skin of the temple, and Taylor[3] from the outer side of the lower lid, the base situated to the nasal side. He made a button-hole vertically through the thickness of the lid near the base of the flap, twisted the flap 180 degrees on its axis, pushed it through the button-hole, and fastened it with sutures through its edges to the lower lid so that the epidermis faced the eyeball.

Similar operations were published by J. J. Chisolm,[4] Snellen,[5] and Browne.[6]

Harlan[7] passed a horizontal ribbon-shaped flap taken from the outer layer of the lower lid, but left it in contact with the skin at both ends, turned upside down, through a horizontal incision through the lid in the fornix region, and stitched the lower border to the inner side of the free margin of the lid. The epidermis faced the eyeball. The external wound was closed by sutures.

Rogman[8] covers the raw surface of the lid with a quadrilateral flap formed from the outer layer of the skin of the lower lid, with its base opposite the lid fornix and its apex towards the cheek. Just below the base he divides the lid by a horizontal section, draws the flap through it, and fastens it by sutures to the *lid*, the apex of the flap to the free margin of the *lid*. The epidermis faces the eyeball. In three or four weeks, when the flap is firmly united to the lid, the sutures and scars along the section of the lid are removed.

A similar skin-flap, formed from the upper part of the lower lid, with

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[1] Stellwag, Rückblicke auf die augenärztlichen Pfropfungsversuche, Allgemeine Wiener medicinische Zeitschrift, 1889.

[2] The Medical Record, 1875, p. 203.

[3] Medical Times and Gazette, July 1, 1876, and Transactions of the Fifth International Ophthalmological Congress, 1876, p. 250.

[4] Virginia Medical Monthly, 1877, p. 180.

[5] The Ophthalmic Review, 1889, p. 384.

[6] The Ophthalmic Review, 1890, p. 236.

[7] The Ophthalmic Review, 1890, p. 351.

[8] Traitement du symblépharon. Nouveau procédé opératoire. Archives d'ophtalmologie, 1892, t. xii. p. 627.

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its apex upward, is drawn through the reopened section in the lid and fastened with sutures upon the *sclerotic*. The epidermis faces the epidermis of the previous *palpebral* skin-flap. This method provides well for the formation of a new fornix.

Samelsohn[1] operates in cases of complete symblepharon of the temporal half of the conjunctiva as follows. The *upper* lid is detached and freed of all cicatricial tissue. The cicatricial tissue on the sclerotic, which is covered with epithelium, is left. A quadrilateral flap, the basis at the margin of the lid, is formed of the outer layer of the skin of the *lower* lid, tilted, and fastened to the raw surface of the upper lid, with loop-sutures tied on the outer surface, and with ordinary sutures on the inner side. The skin-defect is covered by outside sutures. In five days the flap is firmly united to the lid, the sutures are removed, and the basis is cut and can be left to shrink away or used for covering the upper part of the defect in the skin of the lower lid. After some time the adhesion of the lower lid is treated in the same way. The result has been excellent.

Samelsohn thinks that his method may be made available for total symblepharon.

Results.—It is too early to judge of the permanent good the above-described operations may do. Non-pedunculated flaps, apart from the uncertainty of their attachment, shrink so much that they cannot inspire us with reasonable hope. Pedunculated flaps shrink likewise, but not so much. It remains to be seen, first, in what percentage of the cases the operations are a primary success,—*i.e.*, how often and to what extent the transplanted flaps take root and live; secondly, how far the conjunctival sac will be restored, securing the mobility of the eye; thirdly, how far skin can take up the function of a mucous membrane,—in our case, to lubricate the ball-and-socket joint between lids and globe, and keep the cornea transparent. Without any further experience, we can say that skin will never take up the functions of a mucous membrane, and I may say what I said at the beginning of the section: "The value of a symblepharon-operation depends upon the extent of conjunctival surface that is preserved."

VIII. SURGICAL TREATMENT OF TRACHOMA.

The treatment of trachoma depends essentially on the kind, intensity, and stage of the disease in which we find the patient when he presents himself. As to the nature of trachoma, I distinguish, from a clinical standpoint, two kinds of granulated lids or trachoma. The first is represented by patients who, without any or with very trifling symptoms of inflammation or discomfort, have hyaline, sago-like granulations deposited in the retrotarsal fold and on the palpebral portion, none in the fornix and bulbar conjunctiva, though on clinical examination we may suspect the granulations to extend beyond the retrotarsal fold, for when the latter is freed of them by

[1] Operative Behandlung des Symblepharon. Bericht über die XXII. Versammlung der Heidelberger ophthalmologischen Gesellschaft, 1892, S. 149.

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squeezing, the red points, the bloody nests of the granulations, are seen only in the fold, whereas the fornix and bulbar conjunctiva are normal. In many cases the deposition of granules is only moderate, in others so marked that the retrotarsal folds appear like broadened ridges, and the granules are so densely set that they seem to coalesce (diffuse infiltration). Not rarely this form of trachoma is limited to both retrotarsal folds, the palpebral surfaces, and the caruncle of one eye, the other eye being normal in every respect. It is not met with in the lower retrotarsal fold alone, as the follicular catarrh is said to be. Frequently it is diagnosticated only accidentally, the patients, not aware of it, coming for some other complaint. I have called this form *simple* or *non-inflammatory trachoma*. I think it is not contagious. In Europe I have seen it rarely, in New York frequently. In *this form expression celebrates its triumphs*. That it may, however, disappear spontaneously, is attested by Logetschnikof,[1] of Moscow, and exemplified by a case which Greeff[2] depicted and described under the name of pseudo-trachoma.

The other kind of trachoma is the well-known, so-called Egyptian or military ophthalmia. It is both inflammatory and contagious. It undoubtedly is produced by the transmission of virulent secretion from one eye to another. It is endemic and epidemic. It may be the complication and sequel of another kind of conjunctivitis, the catarrhal, blennorrhœal, and diphtheritic. It leads, when left alone, to more or less cicatrization of the conjunctiva and its consequences in the lids, the cornea, and the whole eye. It is not impossible that the non-inflammatory form, by external influences other than contagion, may pass over into the inflammatory form, but I have never noticed it. It seems natural, however, to suppose that an eye affected with simple trachoma is predisposed to inflammatory trachoma.

Indications of Surgical or Mechanical Treatment of Granular Lids.—Non-inflammatory trachoma should be treated by expression as soon as the case presents itself to the physician. In the inflammatory form the acute stage, be it primary or only a paroxysm of the chronic stage, should neither be treated surgically nor with strong chemical means. This period should be tided over by antiphlogistic treatment, cold applications, careful cleansing, and instillation of mild antiseptics. *The mechanical treatment*—expression, spooning, grattage, cauterization, and excision—*is indicated in all cases where the characteristic hyaline granules are present. In almost all cases it should be supplemented by medicinal applications and careful attendance to hygiene.*

Methods.—(1) The *oldest method* of mechanical treatment of trachoma is the ophthalmoxysis, the *brushing* or *scratching* out of the granules, the *brossage* or *scrattage*, revived lately by Borelli (1859), Schröder (1889), Abadie and Darier, of Paris (1890), and introduced into America by

[1] Logetschnikof, Aperçu de l'activité du cercle ophthalmologique de Moscou, 1897, pp. 45 and 52.

[2] Greeff, Archives of Ophthalmology, vol. xxi., 1892, p. 505, and Archiv für Augenheilkunde, Bd. xxiv., 1892, S. 60.

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Marple[1] and Weeks, of New York (1891). With or without previous scarifications, the inner surface of the lid, well exposed by clamping forceps, is firmly scrubbed with a short- and stiff-bristled brush till all the granules are removed. Then a solution of corrosive sublimate—1 : 500 or stronger—is well rubbed into the torn-up tissue. The reaction is relieved by cold applications. The after-treatment consists in antiseptic and astringent washes, or the application of a one per cent. or two per cent. solution of nitrate of silver, or touchings with the sulphate of copper or alum crystal, as customary.

(2) *Galvano-causis* was first used by Samelsohn.[2] He burned the granulations out with a fine loop-electrode.

(3) *Electrolysis* was introduced by G. Lindsay Johnson,[3] of London. The inner surface of the lid, well exposed, is scarified with the "sillonneur," a scalpel with three blades about one millimetre distant from one another. Each application produces three incisions, parallel to the edge of the lid, through which the electrodes are passed.

(4) *Excision of a strip of conjunctiva containing the granules*, first methodically practised by Galezowski,[4] of Paris, has had many warm advocates, especially in Northern Germany and Russia, where trachoma abounds.

Technic.—The conjunctiva is cocainized and sterilized. Professor Schnabel, whose procedure Elschnig[5] describes, uses for this purpose a solution of iodo-trichloride 1 : 2000. The point of a hypodermic syringe, filled with a four per cent. solution of cocaine, is thrust into a fold of conjunctiva (seized and raised near the outer commissure, two or three millimetres behind the convex border of the tarsus) and advanced horizontally the whole length of the retrotarsal portion. In slowly withdrawing the syringe about four minims are injected into the subcutaneous tissue. This produces an artificial œdema, rendering the granules in the raised retrotarsal fold very conspicuous. In ordinary cases a fold from eight to twelve millimetres broad is removed. To limit it, from five to eight threaded needles are introduced vertically through the base of the fold. Their points of entrance are in the healthy bulbar conjunctiva; their points of exit from one to one and a half millimetres in front of the convex border of the tarsus. Having the fold stretched by an assistant, the operator circumcises it with a scalpel about one millimetre inside the needles. Then the end of the fold is held up with forceps and the whole strip excised with scissors. The needles are now pulled out and the sutures tied.

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[1] Marple, The Medical Record, November 28, 1891.

[2] Samelsohn, Die Galvanokaustik in der Ophthalmochirurgie, Graefe's Archiv für Ophthalmologie, Bd. iii., 1, 1857, S. 114.

[3] Johnson, A New Treatment of Chronic Trachoma, Archives of Ophthalmology, vol. xix. p. 264.

[4] Galezowski, Recueil d'Ophtalmologie, 1874, p. 132.

[5] Elschnig, Zur operativen Behandlung des Trachoms, Wiener medicinische Blätter, 1889, Nrs. 14 and 15, and Czermak, Augenoperationen, S. 283.

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In some cases it has been thought advisable to *remove a piece of the tarsus together with the retrotarsal fold.* This can be done in the following way. After the cocaine injection into the retrotarsal fold a longitudinal incision is made through the thickness of the tarsus, but not through the epitarsal aponeurosis. The latter is detached from the tarsus with a round-edged hand-chisel (Fig. 21). The needles are then introduced as above described, but they have to comprise the posterior part of the tarsus. For this purpose they must be passed underneath it,—*i.e.*, between its outer surface and the aponeurosis,—and finally through the conjunctival border of the anterior part of the tarsus. Now the retrotarsal fold and the posterior part of cartilage are removed with scissors, the needles drawn, and the sutures tied.

The *reaction from the operation* is usually moderate, and will be relieved with cold applications. The slight thickening of the wound and of the lid, as well as the suffusion of the latter, disappears in a few days. If the closure of the wound was insufficient or some needles tore through, granulations may sprout from the wound. They should be removed with scissors.

In mild cases the recovery takes between two and three weeks, in severe ones longer.

(5) *Expression* seems to be comparatively a recent method of treating granular lids. Cuignet[1] practised and described it in 1873. Other authors are E. Berlin,[2] Mandelstamm,[3] and Hotz.[4] Dr. Hotz pressed the granulations out with his finger-nails.

In 1891 Dr. Prince sent me a pair of ring-shaped forceps which he used for expression of trachoma, and in the same year I saw Dr. Henry D. Noyes perform expression on a very marked case of non-inflammatory trachoma at the New York Eye and Ear Infirmary. He described the mode of treatment in his text-book. He used forceps the ends of which were grooved and at right angles to the shafts. I used both Prince's and Noyes's forceps a few times, when it suggested itself to me that an instrument acting on the principle of the mangle would express the granulations as well, and with less bruising and laceration of the tissue. Accordingly I had *roller-forceps* made, which I have used since with entire satisfaction. They consist (see Fig. 29) of two creased cylinders, about two millimetres

FIG. 29.

Roller-forceps. (Knapp.)

thick and ten or eleven millimetres long, rolling on pivots in horseshoe-shaped ends at the shafts. Good roller-forceps must roll. To do so the surface of the rollers must offer sufficient resistance (therefore the creases), and there must be sufficient play between the

[1] Cuignet, Annales d'oculistique, t. lxix. p. 78.

[2] E. Berlin, Beiträge zur pathologischen Anatomie der Conjunctiva, Klinische Monatsblätter für Augenheilkunde, 1878, S. 358.

[3] Mandelstamm, Der trachomatöse Process, Graefe's Archiv für Ophthalmologie, Bd. xxix., 1, 1883, S. 100.

[4] Hotz, Treatment of Trachoma by Expression of the Granules, Archives of Ophthalmology, vol. xv., 1886, p. 147; Archiv für Augenheilkunde, Bd. xvi., 1886, S. 412.

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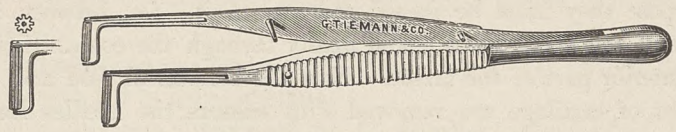
ends of the cylinders and the tips of the handles, otherwise blood and tissue will clog the pivots. The cylinders should not be too thin to give sufficient leverage, nor too thick, like those of Stephenson, which makes the instrument too cumbersome for its delicate work. I have seen two modifications of the instrument, both designed with the object to reach more readily the granulations at the external and internal canthi. The one by Dr. Campbell has the ends of the cylinders projecting beyond the tips of the shaft; in the other, by Dr. Rust (Fig. 30), the horseshoe-formed end-piece is so changed that one arm is in a straight

FIG. 30.



Roller-forceps. (As modified by Dr. Rust.)

prolongation of the shaft, while the other turns off parallel with the cylinder, and leaves space between it and the cylinder. It can, like Noyes's forceps, be pushed easily under the canthi. The instrument works well, and I had one made in which the shafts preserved their horseshoe ends, but these were nearer to the cylinders. The original pattern is probably the strongest and handiest instrument. I find that I can reach with it the recesses in the corners quite satisfactorily.

The cleansing of the roller-forceps requires particular care. The cylinders can be taken out and, as well as the holes in the shaft ends, cleansed separately. This need not be done after every operation, but they should be brushed with soap, boiled, and wiped dry after, and boiled again before each operation.

I demonstrated the forceps and gave the first account of my experience with it at the meeting of the American Ophthalmological Society in 1891.[1]

Technic of Expression of Trachoma Granules with the Roller or other Forceps.—To express trachomatous infiltration both thoroughly and with the proper care not to wound the cornea nor bruise or lacerate the conjunctiva, is painful and requires time. I therefore, as a rule, use general anæsthesia. As the operation has proved harmless, I operate on the four lids at the same sitting. The upper lid when everted should be seized at its edge with the (roller) forceps at the centre of the retrotarsal fold drawn upward, so that the whole extent of the granular deposition shall be exposed. Then the lid is held everted with the index-finger, and one cylinder is pushed deep into the upper fornix over the edge of the lower lid, which, remaining *in situ*, protects the cornea. The other cylinder passes over the tarsal surface of the conjunctiva. The forceps is now compressed with moderate force and drawn forward, so that we notice the even rolling of the cylinder that lies on the tarsal surface. The granules come out more or less crushed by the cylinders, and if they are soft their contents are seen only as a gelatinous liquid. The forceps is introduced and the manœuvre repeated again and again until all trachomatous substance is pressed out. At first the tissue caught between the rollers is thick and resistant, but

[1] See Transactions of the American Ophthalmological Society, Twenty-Seventh Meeting, p. 748.

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gradually it thins down, and when all the infiltrated substance is out the retrotarsal fold stretches as a thin doubled-up membrane between the cylinders. Then the centre of the fold should be drawn up and inward so as to unfold and stretch out the portion concealed behind the outer commissure. This portion is then pressed out with the roller as thoroughly as the central portion. The same ought to be done with the lower lid and the caruncle. If, which is not rare, the tarsal surfaces of the lids are also beset with granules, these can best be pressed out if, on the lower lid, one cylinder passes over the outer and the other over the inner surface. On the upper lid this need not be done, as the longer and thicker tarsus gives sufficient resistance for the roller to liberate also the tarsal surface from the included granules. The impressions from the ridges of the cylinder give to the surface a fluted appearance which must be uniform,—*i.e.*, free from granules. On inspection the whole retrotarsal fold with the canthal portions should be entirely free from granules, and present a dark red surface with a number of small redder dots, apparently the cavities of the granules now filled with blood. The surface may or may not be washed over with a small pad of absorbent cotton dipped in a solution of bichloride of mercury 1 : 500.

Reaction and After-Treatment.—There is no more reaction than slight swelling of the retrotarsal folds, very little subconjunctival hemorrhage in a few cases, no pain, no discharge. The patient, going home immediately after the operation, has nothing to do but to wash and cool his eyes with fresh water several times daily.

The first few days the conjunctival surfaces may be slightly covered with mucus or coagulated fibrin, causing a somewhat wrinkled appearance of the surface, as if there were undue agglutinations. The wrinkles need not be stroked out, for in a few days they smooth themselves. The eyes have only to be washed, and, if there is still some roughness, the lids should be touched with the sulphate of copper crystal, very mildly, three times a week, until the conjunctiva has a normal aspect.

(6) *Curetting* (*excochleation*) *of granular lids*, apparently first mentioned by Bardenheuer,[1] of Cologne, has of late been particularly advocated by Professor Sattler,[2] of Leipzig.

Technic of Curetting.—The conjunctiva is cocainized; in severe cases general anæsthesia is preferable. The capsule of each granule is ripped with a cataract-needle, and the contents scooped out with a small sharp spoon. Spoons of different sizes are necessary, from one to three millimetres in diameter. The tarsus offers sufficient resistance for the needle and spoon, but the retrotarsal folds and the fornix parts do not. These must

[1] Bardenheuer, Indicationen zur Anwendung des scharfen Löffels, Cologne, 1877.

[2] Sattler, Die Trachombehandlung einst und jetzt (The treatment of trachoma formerly and at present), Zeitschrift für Heilkunde, xii., 1891. An excellent paper on the history of the treatment of trachoma. The same subject is also very well treated in Panas's text-book, vol. ii. p. 233, etc.

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be put on the stretch with a fixing forceps, and one granule after the other ripped and spooned.

The sterilization, cleansing, and after-treatment are the same as in the other methods. The reaction is usually slight.

Results and Value of the Different Methods.—We ought to bear in mind that trachoma may be spontaneously recovered from. It seems to me that the non-inflammatory kind should be particularly apt to show such recovery, but I have no proof of it from personal observation. (See publications by Greeff and Logetschnikof mentioned above, p. 847.) I have seen cases of inflammatory trachoma get well without treatment, leaving at least no visible cicatrices. Cases that recover spontaneously, leaving more or less cicatricial tissue, but no functional or other disturbance, are not exceptional.

On the other hand, we know that trachoma is a most rebellious disease, even to treat, and in multitudes of cases leads to permanent impairment of sight, often enough to one-sided or double-sided blindness. Of great influence are the hygienic surroundings. Patients treated for months and months in dispensaries without much benefit rapidly improve when admitted to a well-conducted hospital or sanitarium.

In the second place, we should bear in mind that trachoma has a great tendency to relapse, and that no cure, medicinal or surgical, nor both combined, can give insurance against the possibility of a relapse. Patients should be watched and warned not to lose time if the first symptoms of a relapse occur. Frequently the relapses are very transient, cured by care and a few touchings with the copper crystal.

When the patient comes to us with characteristic spawn-like granulations and no, or no marked, inflammation, especially no blennorrhœa, *surgical treatment is indicated.* The destruction of the granules one after the other with the actual or galvanic cautery, or pressing them out with the finger-nail, or the scooping may be perfectly appropriate, and, supplemented by local chemical treatment, cure the patient well and rapidly enough. These methods are indicated when in the course of the disease the greater part of the conjunctiva has become free from granules and a small number are still scattered and deeply embedded in the tissue.

When, however, there is a multitude of granules embedded in the four retrotarsal folds, so that no free space between them is visible to the naked eye, these methods would be very tedious, if efficient at all. Then *grattage, excision,* or *expression* is indicated. Of these three methods the one is as serviceable and valuable as the others. All three are sufficient for the total removal of the granules, if that be possible, and if not, they may be estimated to be so in the same degree. Each of them is likely to cure the disease rapidly, but the question remains, "Do they cure equally well?" I mean, does the one cure the trachoma with as little damage to the eye as the other? They do not. Grattage and excision cause more destruction of the conjunctiva than expression. As the conjunctiva is an essential part

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of the organ of vision, nothing of it should be sacrificed if we can help it. In favor of excision it may be said that we can spare a strip of healthy conjunctiva without notable discomfort; the same may be said in favor of grattage, though this procedure is less certain as to the limitation of its action. In all three methods we may do too little, and have to operate again. This would be only a loss of time, not the greatest loss the patient could have. We may also do too much,—*i.e.*, attack more than is diseased, which for excision is identical with so much destruction, and for grattage is nearly the same. Grattage, in order to be efficient, produces wholesale tearing up of the conjunctiva, by which a good deal of valuable tissue will be actually destroyed and a certain portion misplaced by the unavoidable cicatrices. Expression is the gentlest surgical method I have become acquainted with. If cautiously done, neither corneal ulceration nor symblepharon follows in its wake. I have, however, distinctly noticed that after the cure of very severe cases the retrotarsal folds are not so extensive as in the normal condition, and there are fine linear scars discoverable in them. Considering the density and depth of the infiltration, it is a wonder that so much conjunctiva was preserved,—*i.e.*, was not destroyed by the granules. When the trachoma granules have disappeared and the conjunctiva is replaced by cicatricial tissue, the mechanical treatment as a curative agent is out of the question. Surgical treatment has to relieve the sequels of trachoma on the lids and the cornea, and to do this as far as possible with the object of restoring lost conjunctiva by procedures which we have discussed above, number vii., among the operations for symblepharon.

In conclusion, I may say that the mechanical treatment of trachoma does not supersede the medicinal,—it supplements it most beneficially in abridging and facilitating the recovery. Expression does this as efficiently, but more gently and with better preservation of the conjunctiva than any of the other methods.

IX. PERITOMY.

Peritomy is an operation which Dr. Furnari, of Paris, later professor of ophthalmology at the University of Palermo, invented and described under the name of *tonsure conjonctivale*. It was used to improve, if not cure, rebellious and advanced trachomatous pannus, and consists in the removal of a strip, from five to eight millimetres in breadth, of conjunctiva around the cornea. It was thought that the vascular cicatricial tissue covering and invading the cornea would waste away if its nutritive supply was cut off. It did not do so. The removal was made deeper. It did not yet answer the purpose. It was made so deep as to destroy all episcleral tissue, and, to be sure that nothing was left, the denuded zone around the cornea was treated with strong caustics. For a time the operation was popular, especially in England. I have performed it a number of times. The result was too uncertain, and the danger of sloughing of the cornea was ever present. I think the operation has been generally abandoned.

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§ VI. OPERATIONS ON THE SCLEROTIC.

There is only one operation done on the sclerotic worth special consideration. It is *sclerotomy*, used chiefly for glaucoma. We distinguish anterior and posterior sclerotomy according as the incision in the sclerotic is before or behind the insertion of the iris.

(*a*) *Anterior sclerotomy* was first performed by Quaglino in 1871, after de Wecker (1867) and Stellwag (1868) had expressed their belief that not the excision of the iris, but the division of the sclerotic, constituted the important factor in the cure of glaucoma by iridectomy. Quaglino published[1] five successful cases of sclerotomy. De Wecker[2] then became very enthusiastic about sclerotomy, L. Mauthner[3] the same. The uncertainty of the results of iridectomy in chronic glaucoma, the numerous relapses after iridectomy, and the occasional occurrences of "malignant" glaucoma, cystoid scars, and other unpleasant conditions, determined many to try sclerotomy.

Indications.—Some oculists preferred sclerotomy to iridectomy in all forms of glaucoma, others reserved it for chronic and simple glaucoma, and for all forms where iridectomy usually fails, in hemorrhagic glaucoma, megalophthalmus, and especially in those cases where after an iridectomy a relapse has occurred; also for glaucoma following an extraction of cataract with iridectomy. I know by experience that these cases are cured by iridectomy, but I do not know whether they are cured by sclerotomy as well. At the present time sclerotomy is less extensively practised than it was ten and twenty years ago.

Technic.—A very narrow Graefe knife is thrust into the anterior chamber one millimetre behind the limbus, pushed through it, and thrown out at a corresponding point on the nasal side. It is best to make sclerotomy upward. The external opening should be from eight to ten millimetres long. The section lies in the plane of the iris, and is made by slow sawing movements, leaving the middle third uncut. De Wecker advises to incise the internal layers of the corneo-scleral border, which is done with the point of the knife while the knife is withdrawn.

Accidents during the Operation.—The pupil may become oval, which is a sign that iris is caught in the wound. We may try to reduce it with a blunt probe. If this attempt does not succeed, or if iris shows in the wound, the iris should be drawn out and cut off.

Course of Healing.—Sclerotomy wounds heal more readily than iridectomy wounds in glaucoma, yet subsequent incarceration of the iris is more than exceptional. This may occasion prolonged irritation of the eye, and not only frustrate a good recovery, but more or less frequently cause permanent damage to the eye. Not rarely the scar is neither smooth nor linear, but

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[1] Quaglino, Annali di Ottalmologia, 1871, p. 200.

[2] De Wecker, Transactions of the Heidelberg Ophthalmological Society, 1869, p. 384.

[3] Mauthner, Archives of Ophthalmology and Otology, vol. vii., 1875, p. 179 (Aphorisms on Glaucoma).

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broad, nodular, and even somewhat raised. De Wecker regards this as a favorable condition. The filtration through the scar, in his opinion, is the efficient element in all glaucoma operations. I have always considered the smooth, even scar as the most favorable, and all others as incarcerations and fistulas. It is true that a fistula covered with conjunctiva is a protection against increased eyeball tension, but so soon as it closes the eye will harden. Uneven scars, as well as small fistulas, may exist for years and give rise to no, or only transient, trouble,—which means a transient closure of the fistula,—but many fistulas sooner or later cause marked pain and hardness of the globe. Only another operation, iridectomy, will render the eye quiet. These cases are difficult to deal with, on account of the visible or concealed incarceration of the iris.

MODIFICATIONS OF ANTERIOR SCLEROTOMY.

Quaglino's sclerotomy consisted in a simple incision with the lance-shaped knife as in iridectomy. He inserted the lance two millimetres behind the transparent margin of the cornea, went obliquely through the lamellæ, as close to the iris as possible, making an inner section of from three to five millimetres, and withdrew the knife very slowly, so as to avoid iris prolapse. Snellen followed Quaglino's procedure.

Landesberg,[1] Bader,[2] and others divided the sclera with a Graefe knife, but preserved the conjunctiva. I have seen a number of Bader's operations in London. The eyes were greatly disfigured by large staphylomas. I could ascertain nothing as to their preservation of sight.

Knies[3] divides, with a Graefe knife, the root of the iris together with the sclerotic.

De Wecker[4] describes the following modification. With a stop-knife (*couteau à arrêt* of his own design), six millimetres broad, he makes an incision into the upper scleral border one millimetre behind the limbus. Then he introduces a pair of iris-forceps no farther than two millimetres beyond the limbus, opens it, seizes the iris near its insertion, pushes the forceps with the seized iris towards the centre of the pupil, lets the iris loose by reopening the forceps, and withdraws the forceps, leaving its branches open. He then tears the iris off its ciliary insertion about five or six millimetres in length, an iridodialysis which earlier oculists used to perform for making an artificial pupil. "An instillation of eserine terminates this operation so easy of execution and so innocent in its consequences."

Panas[5] and de Wecker[6] perform a sclerotomy (*cicatrisotomy* or *oulotomy*)

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[1] Landesberg, Die Anwendung der Sclerotomie beim Glaucom, Graefe's Archiv für Ophthalmologie, Bd. xxvi., 1881, 2, S. 77.

[2] Bader, Royal London Ophthalmic Hospital Reports, viii., 1876, 3, p. 430.

[3] Knies, On a New Treatment of Glaucoma, Transactions of the Heidelberg Ophthalmological Society, 1893, p. 118.

[4] De Wecker, Sclérotomie simple et combinée, Annales d'oculistique, cxii., 1894, p. 261.

[5] Panas, Bulletins et Mémoires de la Société française d'Ophtalmologie, 1883.

[6] De Wecker, La cicatrisotomie ou l'oulétomie, Annales d'oculistique, xciii., 1885, p. 10.

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through the scar of an iridectomy wound when a second operation is necessary on account of a relapse, rather than make another iridectomy opposite the first. A Graefe knife is thrust through the cicatricial tissue from one side to the other, including incarcerated pieces of iris in the corners if there are any. A central bridge is left.

The above modifications are not the only ones proposed and performed. This shows that the various morbid conditions comprehended under the term glaucoma are by no means under control. Universally iridectomy is regarded at present as the general operation for which the other operations are substituted in particular anomalies. When both iridectomy and anterior sclerotomy have failed, another operation is still at hand,—viz.,

(*b*) *Posterior Sclerotomy.*—This operation was first recommended by William Mackenzie, of Glasgow, in 1830, in his classical "Treatise on Diseases of the Eye" and his practical essays on ophthalmology. It should be executed with a broad knife, about seven millimetres behind the cornea, in the lower half of the globe. The knife should be advanced towards the centre of the vitreous, and slightly turned on its axis for one or two minutes to let some liquid escape.

De Luca published[1] the same operation in 1872, without knowing of Mackenzie's recommendation. He made it in almost or wholly blind eyes to relieve pain.

Posterior sclerotomy has been done in three varieties:

(1) As a simple *paracentesis.* Shorter or longer incisions through the sclera were made, mostly in the lower segment, between the ciliary muscle and the equator, and the external and inferior recti. Their object was to *relax the eyeball capsule when distended by serum or blood.* As indications were mentioned *megalophthalmus, glaucoma* (especially absolute, to relieve pain), *hæmophthalmus,* and *detachment of the retina.* Owing to the permeability of the choroid and retina, liquids may escape in this way from the vitreous as long as the wound in the sclerotic remains pervious. This perviousness could be increased and prolonged by an angular cut (⌜ or ⊤), such as Parinaud has made through the sclerotic and the deeper coats. As to the results of these attempts I know nothing definite and reliable to cite.

(2) The *usual posterior sclerotomy,* which is a *sclero-chorio-retinotomy.*

Technic.—A broader or smaller (Beer's or Graefe's) knife is plunged into the vitreous through the lower outer quadrant of the eye, between the external and inferior recti, behind the ciliary body. The incision should be five or six millimetres long, and not penetrate deeper into the vitreous than one centimetre, unless it is intended to puncture detached retina.

There is no particular accident during the operation to be noted, and the after-treatment is simple. The eye is bandaged, and the patient should keep his bed for several days. The wound may be sutured or not, as the case seems to indicate. The healing is commonly smooth.

[1] De Luca, Sulla Paracentesi della Sclerotica nel Glaucoma, Annali di Ottalmologia, t. ii., 1872, p. 155.

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(3) *Sclerocyclotomy* (*Hancock's Operation*).—Forty years ago, Hancock, ophthalmic surgeon to Charing Cross Hospital, acting on the supposition that glaucoma was the result of a strain or spasm of the ciliary muscle, invented this operation. It consisted in a division of the ciliary muscle. He entered the eyeball with a straight lance-shaped knife about three millimetres behind the limbus corneæ, on the outer side, obliquely from below and down to above and back, thus dividing the ciliary muscle in its whole extent, without injuring the lens. There were no particular accidents, and no striking results. Hancock had some followers, but his method of operation has not stood the test of time.

INDICATIONS AND RESULTS OF POSTERIOR SCLEROTOMY.

(1) In *traumatic hæmophthalmus*, without rupture of the corneo-scleral capsule, it may be beneficial, as this injury is not infrequently followed by increase of eyeball tension and its consequences. Yet in hemorrhages of that kind there are commonly such extensive lacerations of the uveal tract and the retina that sight will remain greatly impaired. Nevertheless I have seen cases when the patients remained blind for many months, then the blood, which had even stained the cornea, gradually absorbed, and good sight was restored. If this can be done by nature, art may abridge the disease and ward off consequences for which the duration of the disease is responsible. The indication for sclerotomy, either scleral or deeper, will chiefly depend on the presence of increased eyeball extension.

(2) In *glaucoma*, posterior sclerotomy is indicated when iridectomy and anterior sclerotomy have failed to relieve the patient,—*i.e.*, in absolute and so-called malignant glaucoma. This indication is strongly upheld by French oculists. I have seen good results from it in a few cases of my own practice.

(3) In *detachment of the retina*, sclerotomy with puncture of the retina, as is well known, was first practised by v. Graefe, who was led to it by cases of detachment in which the retina was reattached when it had ruptured. Retinal paracentesis has been practised in all varieties alone and in combination with many other modes of treatment, each of which has one or several cases of recovery in its support. The operative treatment of detachment of the retina would require a chapter for itself; at present authentic facts are not available in sufficient number to undertake such a work. There are two great facts: detachment of the retina is cured in a limited number of cases; it is not cured in the great majority. There is not yet any operative procedure that has sustained by facts its superiority over other modes of treatment. A well-read and critical observer, unprejudiced by the experience and claims of others, basing his judgment on the cases that have come under his own care, will, like the present writer, probably come to the conviction that rest of body and eyes is of paramount importance in the cure of retinal detachment. There are certain forms of detachment of the retina that recover more readily than others; for instance, the trau-

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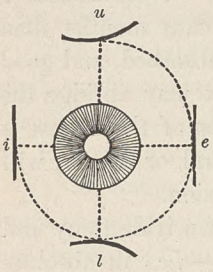
matic and the choroiditic. Retinal detachment is only a symptom, just as dropsy is. We have to study the fundamental disease and select the remedy accordingly, be it hygienic, medicinal, or operative.

(4) Sclerotomy posterior is an *initial step to other operations;* for instance, the removal of intra-ocular parasites, especially cysticercus, in which our German colleagues are so expert, or the removal of foreign bodies from the vitreous and the background of the eye, either by surgical instruments or by the magnet.

§ VII. OPERATIONS ON THE MUSCLES OF THE EYE.

As the operations for anomalies of motility—strabismus, insufficiency, and paralysis—have to *solve optico-dynamic problems mechanically*, a few introductory remarks on the topographical anatomy of the tendons of the four recti muscles may be appropriate. The line of insertion of the tendons of the four recti muscles (Fig. 31), according to the investigations of Motais, verified by Panas,[1] resembles a spiral, the distances of their centres from the cornea being as follows: rectus internus (Fig. 31, *i*), 5; inferior (*l*), 6; externus (*e*), 7; superior (*u*), 8 millimetres. Arlt gives rectus internus, 5.5; externus, 7.5; inferior and superior, 6.5 each. There are individual variations; but, as far as I can judge, Motais is nearer to correctness than Arlt. The length of the insertion lines is from ten to eleven millimetres; that of the inferior rectus only from nine to ten millimetres. The lines of insertion of the internal and external recti are perpendicular to the horizontal meridian; that of the inferior rectus, and still more that of the superior rectus, are somewhat convex towards the cornea, with a stronger backward curvature at their temporal ends,—facts to be borne in mind. The tendons are surrounded by Tenon's capsule, which forms a serous cavity enveloping the whole sclerotic and supplying each muscle with a sheath, and sends connective-tissue fibres through the orbit to the periosteum by which the eyeball is suspended. These fibres are stronger and more numerous over the tendons of the muscles, and expand as they approach the orbital wall. They limit the rotation of the eyeball, and are called check-ligaments by English and *ailerons connectifs* (connecting wings) by French authors.

FIG. 31.



Insertion of the four recti muscles of the left eye; the line of insertion around the cornea has the form of a spiral: *i*, internal rectus, 5 mm. distant from the cornea; *l*, lower rectus, 6 mm.; *e*, outer rectus, 7 mm.; *u*, upper rectus, 8 mm. The tendons of the lower and upper recti have curved lines of insertion; their posterior ends curve strongly backward.

The operations on the muscles are intended to move the insertion of the muscle either backward (tenotomy) or forward (advancement, prorrhaphy).

Strohmeier, of Hanover, recommended tenotomy of a muscle for the cure of strabismus in 1838, and Dieffenbach made the first squint-operation (a myotomy) in 1839. The

[1] Panas, Traité d'Ophtalmologie, t. ii. p. 2.

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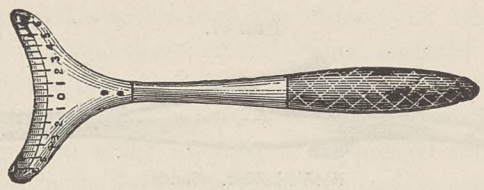
operation was soon overdone, and fell for a time into discredit. It was perfected in its technic by J. Guérin[1] and Bonnet[2] (who made subconjunctival *tenotomies*, not myotomies), Ruete, Boyer (1841), Böhm (1845), A. von Graefe[3] (1853), and others.

Böhm urged the division of the tendon close to the sclerotic; F. Cunier introduced the conjunctival suture; Jul. Guérin introduced advancement; Critchett improved the operative procedure of advancement; A. von Graefe entered thoroughly into the theory of strabismus and improved the operative methods. He taught the diagnosis of insufficiency and cured muscular asthenopia resulting from insufficiency of the interni by tenotomy of the externi. George T. Stevens, of New York, extended our knowledge of the insufficiencies by recognizing, and operating for, muscular asthenopia and its reflex disturbances when resulting from other ocular muscles. He introduced a comprehensive nomenclature for the different kinds of insufficiency. Dr. Savage, of Nashville, has been at work to unravel the disturbances resulting from affections of the obliques.

The *instruments* necessary for squint operations are:

(1) A strabometer. (Fig. 32.)

Fig. 32.



Strabometer.

(2) A wire speculum. (Fig. 2.)

(3) Two pairs of fixing forceps, one for drawing the eye to the side, and steadying it if the patient has it not sufficiently under control, the other for raising the conjunctiva. (Fig. 3.)

Fig. 33.

Delicate, straight, toothed forceps. (Stevens.)

(4) A pair of delicate, straight, toothed forceps (Stevens's) for raising a fold of conjunctiva and tendon in the button-hole operation (of Snellen). (Fig. 33.)

Fig. 34.

Curved strabismus-scissors.

(5) Curved strabismus-scissors, the points small, yet blunt, so as not to pierce the sclerotic. (Fig. 34.)

[1] J. Guérin, Annales d'oculistique, xxii., 1849.

[2] Bonnet, Traité des sections tendineuses dans le strabisme, 1841.

[3] Von Graefe, Archiv für Ophthalmologie, iii.

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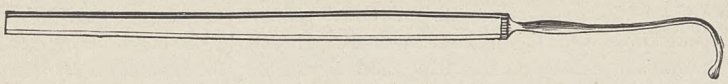
(6) Two larger squint-hooks (Fig. 35) for the ordinary tenotomy, and two smaller and thinner ones (Fig. 36) for the button-hole operation.

Fig. 35.



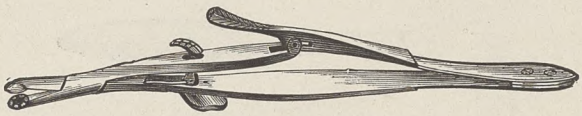
Large squint-hook. (Graefe.)

Fig. 36.

Small squint-hook. (Stevens.)

(7) Needle-holders. (Fig. 37.)

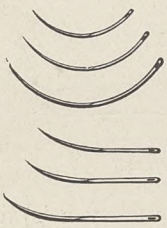
Fig. 37.



Needle-holder. (Sands.)

(8) Fine curved and half-curved needles (Fig. 38) and threads. I prefer a Sands needle-holder with this modification for the use of curved

Fig. 38.



Fine curved and half-curved needles.

Fig. 39.

Needles with spring-eyelets.

needles that the closing surfaces of the tips of the branches are not plain, but the one concave, the other convex; both curved with the same radius as the needles. In this way the holder will grasp the needle firmly with less risk of breaking it than if the opposing surfaces of the holder are plain and the needle between them is curved.

(9) Needles with spring-eyelets (*aiguilles à ressort* of Wulfing-Lüer). (Fig. 39.) They are very easy to thread, and particularly convenient if a needle breaks when introduced into the second lip of the wound, after having safely passed the first, an annoyance not infrequent in advancement operations.

We should use two kinds of thread (black China bead silk), the one very thin for the conjunctiva, the other a little heavier for the muscle and

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sclerotic in cases of advancement. Catgut may be used when not subjected to great traction.

Preliminary Examination.—Before an operation is decided upon, the following points should be established and well considered with regard to their influence on the final result, as well as on the choice of the operative method.

(1) The *direction and kind of the strabismus,—i.e.,* whether convergent, divergent, etc.; unilateral or alternating; continuous or periodical; constant or changing in degree; comitant (= concomitant) or paralytic.

(2) The *history* and duration of the strabismus, including former treatment and its effect.

(3) The results of the *physical examination* of both eyes: cornea, lens, media, background, especially the yellow spot.

(4) The results of the *functional examination of both eyes:* vision, field, refraction, positive and negative binocular vision,—*i.e.,* does the patient see double without being questioned, or only when specially examined for it by red glasses, etc.; is binocular vision discoverable in the whole field of fixation (*Blickfeld*), or only in a part of it; where situated and how extensive is the region in which diplopia is suppressed for all methods of examination (defect in the field of fixation, suppression area).

(5) *Degree of strabismus,* linear and angular (by the perimeter-candle or some other test), with and without correction of ametropia (4 mm. = 20°–25°, 9 mm. = 45°, about the normal adduction limit).

(6) *Power of the muscles* in every direction of the field of fixation,—*i.e.,* abduction, adduction, sursumduction, and deorsumduction (Tropometry).

(7) *Movement of the near point of convergence,—i.e.,* the crossing point of the visual axes, when the sign of fixation is moved towards or away from the eye.

The latter two points are of importance in estimating beforehand the effect of a tenotomy. A weak antagonist will have a diminishing influence, the same as a constant or even an increasing degree of strabismus when the object of fixation is approached; whereas a strong antagonist and a diminishing degree of the strabismus when the object of fixation is approached have an increasing influence on the effect of an otherwise equal tenotomy.

(8) The habitual position of the head, especially a *compensating turning of the head to the side of the antagonist.*

(9) *Protrusion of the eye* and enlargement of the palpebral fissure.

Special preparations are not required. Tenotomy is a short and not very painful operation. I prefer to cocainize the eye only superficially, a few drops of a four per cent. solution to be instilled in the region of the operation from three to five minutes before the operation. Thorough cocainization renders the conjunctiva and episcleral tissue so anæmic and inelastic that it is somewhat difficult to pick up the conjunctiva, Tenon's capsule, and the tendon. Moreover, the absence of bleeding is unfavorable, depriving

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the tissue of the irrigation with blood, the natural and best antiseptic. General anæsthesia (ether) should be administered to children and nervous persons even for ordinary tenotomies, and to most persons for advancement operations, for these require more cutting and stitching, which, apart from the cocaine, would by itself render the muscle hard and stiff.

(*A*) TENOTOMY OR SETTING THE MUSCLE BACK.

There are *different methods of tenotomy* which all yield good results. From personal experience I can recommend the following four plans.

(1) *Arlt's Method.*[1]—The conjunctiva is grasped in front of the insertion of the tendon with fixing forceps (one branch up, the other down), raised in a fold, which is incised with squint-scissors, so as to make an aperture of from six to seven millimetres. The branches of the forceps are now closed and moved two or three millimetres backward over the tendon, then opened from seven to eight millimetres vertically to the sclerotic, to grasp and raise the tendon near its insertion. The tendon is detached with strabismus-scissors from one end to the other as near to the sclerotic as possible. A small squint-hook, introduced under the detached tendon, should pass unrestrained over the insertion. Arlt gives some very useful details in making this examination, which, being common to all methods of tenotomy, I shall here transcribe. The blunt (strabismus) hook should be used as a probe, introduced immediately behind the line of insertion of the tendon, its point held down upon the sclerotic, passed up just beyond the border of the tendon, and moved towards the cornea. If it is stopped some fibres have been left, which must be cut. The same is done downward. Pushing the hook too far upward engages it in the capsule, moving it too far backward and holding the point away from the sclerotic engages it in the muscle, and the cellular and vascular tissue, without finding the stray fibres.

The wound is closed with sutures when by examination the effect is found satisfactory. See below.

(2) *Von Graefe's Method.*[2]—*Dividing the tendon when raised with a hook and fully exposed.* The conjunctiva is incised between the cornea and the insertion of the tendon, and dissected backward and downward beyond the lower border of the tendon. A strabismus-hook is then introduced between the conjunctiva and the sclerotic with the tip down, and turned in such a way as to push the tip of the hook up under the muscle until it comes out above the upper border of the latter; the conjunctiva is moved from below upward with the end of the closed scissors beyond the tip of the hook that projects at the upper border of the tendon. The latter, fully exposed and slightly raised with the hook, is detached with the scissors close to the sclerotic. After ascertaining that the tendon is completely detached and the desired effect obtained, the wound is closed with a conjunctival suture.

[1] Arlt, Operationslehre, Graefe-Saemisch, Handbuch der gesammten Augenheilkunde, Bd. iii. S. 398.

[2] Von Graefe, Beiträge zur Lehre vom Schielen, Archiv für Ophthalmologie, iii. 1857.

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(3) *Critchett's Method.—Subconjunctival tenotomy.* The subconjunctival tenotomy, inaugurated by Jules Guérin and Boyen, was particularly developed by George Critchett, of London.[1] The present writer, after having often seen it done by Critchett himself, has, as a rule, performed it in the following way.

A pair of fixing forceps is pressed, closed, on the conjunctiva, directly over the lower part of the insertion of the tendon,—*i.e.*, for one of the lateral muscles a little above a line drawn horizontally through the lowest point of the corneal margin. In order to strike that point while operating we have to keep the position of some conjunctival vessel in mind as a landmark. This is particularly important in children, who, be they anæsthetized or not, will roll their eyes so that, without a mark, we are apt to make the incision too much below. This mistake is, however, best obviated if an assistant draws the eyeball directly downward. The forceps are opened vertically four or five millimetres, and immediately, under continued pressure on the sclerotic, closed again, so that not only the conjunctiva but also the subconjunctival tissue and Tenon's capsule are grasped and raised together in one small fold. This fold is incised with strabismus-scissors by two cuts, the first dividing the conjunctiva, the second Tenon's capsule. The scissors should not be withdrawn between the first and second cuts, but simply reopened and pushed on. When on removing the scissors we slightly raise the fold, Tenon's capsule shows as a sharply defined sickle-shaped membrane over the sclerotic. The scissors should not be pushed in too deeply, lest the opening be unnecessarily large and orbital fat protrude from it. While the forceps keeps the fold raised, a strabismus-hook is introduced through the conjunctivo-capsular opening under the tendon and pushed upward until the whole, or at least the greater part, of the tendon lies upon it. The forceps is removed, the tendon raised with the hook, and one branch of the scissors pushed behind the tendon, the other in front of it, gently burrowing through the subconjunctival tissue. The tendon is then divided close to the sclerotic in two or more cuts, during which the scissors need not be withdrawn. When the whole tendon is severed the hook can be moved without resistance to the cornea. If this is not the case, we must reintroduce the scissors and cut the fibre-bundles that escaped before. To ascertain whether the whole tendon is detached from the sclerotic, we pass the point of the hook from behind the tendon forward towards the cornea above and below. Any fibres left will offer a firm resistance. We should avoid going too high above the tendon, for in that neighborhood we not infrequently meet with a larger vein, offering almost as great a resistance as a remaining bundle of tendon fibres.

(4) *Snellen's Method.*[2]*—The conjunctiva and tendon are button-holed near*

[1] G. Critchett, Medical Times and Gazette, 1857.

[2] Snellen, Klinische Monatsblätter für Augenheilkunde, Jan., 1870; described also in Knapp's Travelling Notes, Archives of Ophthalmology and Otology, vol. ii., No. 2, 1871, p. 169.

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the centre of the insertion of the tendon, and the tendon is detached upward and downward. Snellen takes up and incises vertically or horizontally a fold of conjunctiva near the centre of the insertion of the tendon from two to four millimetres in extent. He then seizes and pulls up a small portion of the tendon, incises it vertically, passes a delicate squint-hook upward through the hole in the tendon, and divides the upper half of the tendon subconjunctivally with a delicate pair of scissors. Then he divides the lower half in the same way. The conjunctival wound is sutured. George T. Stevens has introduced Snellen's method in America, and devised scissors, which are excellent for the purpose (see Fig. 11, text-plate facing page 785), combining strength with delicacy.

In the description of the above methods I have supposed that the *internal rectus* is tenotomized in a simple case of convergent strabismus. The centre of the incision is in the adult about five millimetres behind the corneal margin, and the section is straight upward. Tenotomy of the tendon of the external rectus is done in the same way, only we must bear in mind that the insertion of the tendon is seven millimetres behind the margin of the cornea. In tenotomy of the inferior and the superior rectus we have to regard not only differences in the distance from the cornea at which the conjunctiva should be incised, but also differences in the direction of the tendon. The centre of the insertion of the tendon of the inferior rectus is six millimetres distant from the cornea; that of the superior rectus, eight millimetres. Furthermore, the direction of the insertion of the tendon is not at right angles to the vertical meridian, but cuts it at an angle of twenty degrees, and its temporal end curves backward. (See Fig. 31, p. 858.)

It is, therefore, proper to attack the tendon not from the temporal side, but from the nasal.

EXAMINATION OF THE EYE IMMEDIATELY AFTER THE TENOTOMY.

If the patient is under the influence of an anæsthetic, we have to wait until this period is past before we examine the eyes. In patients that have not taken an anæsthetic we may examine the eyes immediately or leave it until later. In every case it is advisable to let half an hour elapse before the examination, so that both the mind and the eye of the patient shall be quiet. The *first point* to examine is *how much the deviation has been reduced.* The effect of an ordinary tenotomy varies, according to my experience, between three and five millimetres for the internal rectus; for the external rectus the average effect is less and varies between smaller limits,—namely, between two and three and one-half millimetres. The tenotomy of the superior or inferior rectus has an effect of from two and one-half to four millimetres. The linear measurement is accurate enough for ordinary squint cases, but it is not so for cases of insufficiency, as will be detailed hereafter.

The *immediate effect commonly differs from the ultimate effect,* a fact which has to be taken into account when we ascertain the immediate effect of the operation.

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(*a*) After a tenotomy of the *internal rectus* the primary effect is apt to diminish for several days, but then, as a rule, it increases gradually for weeks, months, and years. In many cases we rejoice at having obtained a perfect cosmetic result, and when we see our patient again in later years his eyes diverge more or less. This brought the squint operation into bad repute, and we have most scrupulously to guard against such an occurrence.

As *a rule, no squint operation should weaken the muscle beyond its physiological limit of power.*

After a *tenotomy of the internal rectus, the result of the immediate examination should be as follows.*

(1) In adduction the medial margin of the cornea should readily reach the caruncle.

(2) The near point of binocular fixation should not be less than five centimetres (two and a half inches).

(3) The eyeball should not protrude.

(4) There should be some convergence left.

As failure in any of these four points is of itself sufficient to throw the eyes afterwards into divergence, we should at once reduce the effect of the operation so that the correct position and mobility are secured. If none of these points is wrong, and the patient shows the full effect of an ordinary tenotomy of the internal rectus,—*i.e.*, four or five millimetres,—the wound is closed with a simple conjunctival suture.

In many cases no fault can be detected in any of these points and yet the patient still squints. If the deviation is only two and a half millimetres or less, we may at once reduce it, but if it is sufficiently large to warrant a tenotomy on the other internal rectus, this should be done later, of which more hereafter.

(*b*) The immediate effect of a tenotomy of the *external rectus* increases the next three or four days, then it diminishes very gradually, so that the ultimate result is frequently not more than two or three millimetres, and at times it disappears altogether. In divergent strabismus we need not fear to throw the eyes into convergence, but in cases of insufficiency of convergence (exophoria) this may occur.

(*c*) As far as my experience goes, the immediate effect of a tenotomy of the *superior rectus* increases, sometimes considerably, during the next days and weeks, then it diminishes, but not so much as with the external rectus. In exceptional cases it disappears altogether.

(*d*) My experience in tenotomy of the *inferior rectus* is not extensive. I can say that the immediate effect of it is greater than that of the external rectus; that it increases for some days, then diminishes again, yet with it, as with the superior rectus, a considerable effect remains permanent, smaller than with the internal rectus, but greater than with the external.

The examination immediately after the operations on the superior and inferior recti should be conducted on the same principles as the examination of the internal rectus. The points to be determined are not so precisely

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fixed as with the latter muscle, yet the examination can and should be conducted with sufficient accuracy to base on it the immediate correction and obtain a satisfactory permanent result. The loss of mobility must not be great; if the eye lags over one or two millimetres behind the other in elevation or depression, if in the median plane for an object of fixation held from fifteen to twenty-five degrees below the horizon and about forty centimetres away from the eye there is still a notable deviation, especially if the eyeball protrudes, the effect should at once be diminished.

MEANS OF CORRECTING THE IMMEDIATE OPERATIVE EFFECT.

(*a*) PROCEDURES TO DIMINISH THE EFFECT.

The most useful and the only reliable, but also perfectly satisfactory, procedure is the *application of sutures*. If we stitch the edges of the conjunctival incision together, we close the wound without influencing the effect (simple *closing suture*), but by applying the sutures in a different way we can produce any degree of diminution of the operative effect, and even annul it altogether (*restrictive suture*). Any restrictive suture to be reliable must have a firm and durable hold on the corneal side of the wound,—*i.e.*, it should pass through the superficial layers of the sclerotic; the insertion of the tendon can be utilized for this purpose. The other side of the suture goes not only through the conjunctiva but also through the tendon or muscle. One branch of a broad fixing forceps is passed backward through the wound, the other over the outer surface of the conjunctiva, so that both conjunctiva and muscle are held together in the grasp of the instrument and drawn forward. If we want a small correction, we pass the curved needle only through the anterior portion of the tendon and through the projecting line of insertion of the tendon and the adjoining layers of the sclerotic. If we want a larger correction, we pass the suture farther back through the tendon or muscle and through the sclerotic nearer to the cornea, and close it only temporarily after the first (double) twisting of the thread, drawing it tighter if the effect is insufficient, or loosening it with a fine squint-hook if it is too great. It is prudent to leave a certain degree of convergence, which will diminish when the suture is cut. If in the next days the effect is still decidedly too great, the suture may be cut; if not too great, it may be left longer. If, also, after cutting the suture the convergence appears too great, a delicate squint-hook is introduced into the wound, and the tendon, from the centre to the borders, loosened from the sclerotic more or less until the position and mobility of the eye are satisfactory.

We may apply more than one suture, and draw forward that part of the tendon and Tenon's capsule which has been set back more than we wished.

Should we have cut the restricting sutures too soon, we can reapply them a day or two later. From sufficient experience in this line I can state that during the first week after the operation the connection of the divided

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tendon with the sclerotic is loose enough to be broken with a hook and either set back or forward without danger of inflammation.

(*b*) PROCEDURES TO INCREASE THE OPERATIVE EFFECT.

(1) *Extension of the Wound Up and Down.*—We examine with the squint-hook the resistances on the upper and lower ends of the line of insertion of the tendon, and loosen them with the hook if they are weak, but if they are strong and the convergence is still very marked, we cut into them with the scissors. This should be done most cautiously, examining the effect after each cut, as the division of the lateral sheaths of the tendon weakens the check ligaments, and lets the muscle retract more, not only immediately but long after the operation. This, in fact, is probably the cause of the slow development of strabismus divergens. It also makes the eyeball protrude and the caruncle sink, giving the eye the staring look we not infrequently see after squint operations.

(2) Application of suture in the conjunctiva over the tendon of the antagonistic muscle (von Graefe). Conjunctival sutures have very little, if any, appreciable effect.

(3) *Drawing the eyeball to the other side by a suture passed through the superficial layers of the sclerotic near the cornea and the corresponding lid commissure* (Knapp). In case of the tenotomy of the internal rectus, a needle is thrust through the superficial layers of the sclerotic near the cornea on the temporal side and then through the thickness of the lid near the outer canthus. By tying the ligature the eyeball is drawn to the outer corner and kept there a day or two, until the tendon has taken its new insertion at the sclerotic.

In case of tenotomy of the external rectus where a greater effect is desirable, one needle of a double-armed thread is put through the superficial layers of the sclerotic near the medial margin of the cornea. Both needles are now passed through the inner commissure over the caruncle, two millimetres apart, the two ends are pulled towards the nose until the medial edge of the cornea touches the inner commissure, the threads are tied, and the eye is kept in this forcible adduction one or several days. Although the divided tendon is in this way held farther back during the time its new insertion takes place than it would otherwise be, the ultimate effect of this procedure is only moderate.

(4) Jocqs[1] *unites the conjunctiva of the eyeball* (of which he takes up two folds near the cornea with a doubly armed thread) *with the conjunctiva and subconjunctival tissue of the lid* near the external canthus by passing the needles through it. With this suture he can draw the eyeball forcibly outward. I have not used or seen this procedure.

None of these methods should increase the effect of the tenotomy beyond

[1] Described by Lagrave, Contribution à l'étude du traitement chirurgical du strabisme, Thèse de Paris, 1893.

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the allowable maximum, as stated before (page 865). If in spite of a correct tenotomy followed by its maximum effect the strabismus is only partially removed, a tenotomy of the corresponding muscle of the other eye, or an advancement, either muscular or capsular, of the antagonist of the same eye, should be resorted to. The latter may be done in the same sitting, the former either soon or long after it, when optical and expectant treatment shall have been tried long enough to make recovery by further non-operative treatment quite improbable.

ACCIDENTS AND MISTAKES DURING THE OPERATION.

(1) We may *operate on the wrong eye.* When the squint is alternating or the patient anæsthetized, the squinting eye may not differ from the other. In the first case it does not matter, in the second it may be a mistake. We should always operate first on the weaker and more crooked eye, or, in other words, tenotomize the muscle which has the greatest contracture. To prevent mistakes, I am in the habit of giving the patient a memorandum of the result of my preliminary examination, and additionally examine him again cursorily immediately before the operation, to verify the statement derived from my previous examination.

(2) *Hemorrhage.*—In exceptional cases there is inordinate hemorrhage, which will fill Tenon's capsule so that the eye protrudes. This is mostly owing either to an abnormal vascular condition or to cutting too deeply into the orbital cellular tissue, after which even *small lumps of fat* may show in the wound. The fat should be excised, and in the case of profuse hemorrhage the operation should be interrupted and a compressive bandage applied. I have never been obliged to leave a tenotomy unfinished on account of hemorrhage.

(3) *Perforation of the Sclerotic.*—This accident is rare, but may occur to any operator who uses the squint-hook.

About ten years ago Dr. H. Derby[1] reported before the American Ophthalmological Society two cases of perforation of the sclerotic during a squint operation which had come under his observation. When he sat down the present writer rose and said that he had met with this accident three times in his own practice, and briefly related the cases. This loosened the tongues of others, and it came out that many of the older men had had the same mishap.

In my own cases it occurred by using strabismus-scissors which had been newly sharpened, so that the points were like daggers. The scissors were handed to me by the assistant, and I had omitted to examine them. Dr. Czermak says correctly that the accident happens when the axis of the scissors is directed somewhat obliquely from behind towards the insertion of the tendon, to which there is a tendency when, standing in front to the right side of the patient, we detach the tendon of the right internal or the

[1] H. Derby, Penetration of the Eyeball, etc. Transactions of the American Ophthalmological Society, vol. iv. 1885, p. 33.

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left external rectus, the deeper branch of the scissors then working into the sclerotic. He advises always to make the section a little from behind forward. I think the axis of the scissors should be parallel to the insertion line of the tendon, because this detaches the tendon most cleanly. The points of the scissors should be blunt, but, as stated above, not broad.

If this accident occurs we may finish or delay the tenotomy, bandage the eye, and treat it as we treat an eye on which extraction has been performed. My own cases recovered without disturbance and were successful.

AFTER-TREATMENT AND COURSE OF HEALING.

It is prudent, though not absolutely necessary, to keep the eye bandaged for a day or two, not at night, but during the day, and not use the other eye, but hang a patch over it, to be raised only during the meals. There is usually no pain during the process of healing. The blood under the conjunctiva absorbs in one or several weeks; if it is retained in larger quantities, it requires a longer time, but leaves no ill effects; the exophthalmus also disappears.

Very rarely *infection* and more or less *intense suppurative inflammation* occur. Arlt says that infection occurs more frequently after subconjunctival than after open tenotomy, and he may be right, for the subconjunctival tenotomy is a close parallel to the inoculation procedures of bacteriologists. It is not to be wondered at that every now and then pathogenic microbes are transported into the wound with the squint-hook, for they are constant tenants of the conjunctival sac.

Five years ago it happened to me that a subconjunctival tenotomy in a scrofulous girl was followed by nodular conjunctivitis, which did not prove to be trachomatous, but tuberculous, the tubercle bacillus having been found by inoculation and the microscope. I destroyed the nodules with the galvano-cautery, and had the patient instil a solution of bichloride of mercury (1 : 5000) in the conjunctival sac every hour or two. During the first month I took her to the hospital, where she was in good hygienic conditions. The inflammation disappeared with the nodules in six months. I saw the patient again eight months ago. She was well, and both her eyes were healthy and straight.[1]

I have seen a few milder cases of suppurative inflammation, and one severe one, in my own practice. The severe one was a boy who immediately after the operation in winter was taken home, thirteen miles away, in a carriage. He vomited repeatedly. The inflammation extended to the neighboring cornea, and left a small corneal opacity, with excessive divergent strabismus, which a number of years later I succeeded in curing perfectly by an advancement.[2]

Once I lost an eye after a simple tenotomy on a child. The operation

[1] See the full account in the Jubilee volume in honor of Professor Helmholtz at his seventieth birthday, 1891. "Festschrift zur Feier des siebzigsten Geburtstages von H. v. Helmholtz." (H. Knapp, Beitrag zur Tuberculosenfrage, S. 28.)

[2] See full account in Archives of Ophthalmology, vol. xv., 1886, p. 436; Archiv für Augenheilkunde, Bd. xvii., 2, S. 158.

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was smooth, but three days later the patient had a violent outbreak of scarlet fever with diphtheria, which was epidemic in New York at that time. The tenotomy wound became diphtheritic, and the eye was lost by panophthalmitis. The child was operated on during the period of incubation of scarlet fever. This, of course, could not have been foreseen. Two other children were operated on in the same afternoon at the same time with the same instruments and at the same place (the clinic). Their eyes showed no reaction.

Other cases of severe reaction have been reported: tenonitis, by Pooley,[1] Wecker,[2] and others; orbital phlegmon with atrophy of optic nerve, by Haase;[3] detachment of the retina, by Mooren;[3] essential phthisis, by Nagel;[4] hemorrhage in wound and eye for eight and a half months with total blindness in a hæmophile, by Ottava;[5] suppuration of sclerotic and panophthalmitis, by von Graefe.[6] I have seen several cases of severe tenonitis in consultation. The eyes recovered, but mostly had divergent strabismus afterwards.

All this shows that a tenotomy is not altogether a harmless operation, and that a prudent ophthalmic surgeon carefully inquires not only into the optico-dynamic condition of the eyes, but also into the general health and the surroundings of the patient, before he operates.

VARIETIES OF TENOTOMY.

Of these I mention only the following.

(1) V. Hasner[7] directs the patient with convergent strabismus to look outward as much as possible. At the same time he presses with a half-opened pair of fixing forceps on the conjunctiva and sclerotic immediately behind the insertion of the tendon, seizes and raises conjunctiva and tendon, and cuts them with a pair of curved scissors. The operation is done in a few seconds. If a few fibres are left uncut, they are found and raised with a hook, and divided with forceps.

(2) E. Gruening,[8] of New York, in consideration of the smallness of the effect in tenotomies of the external recti in divergent strabismus, divides both tendons or muscles (the farther back from their insertions the greater the divergence is). He then puts threads through both tendon-stumps, and unites them over the nose, thus drawing the eyes forcibly inward. He obtains large effects from this procedure.

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[1] Pooley, Archives of Ophthalmology and Otology, vol. iv. p. 410.

[2] Wecker, Traité des maladies des yeux, vol. ii. p. 1045.

[3] Haase, Archiv für Augenheilkunde, Bd. ix., 4, S. 442; Archives of Ophthalmology, vol. ix., 1880, p. 317.

[4] Nagel, Archiv für Ophthalmologie, Bd. xiii., 2, S. 407.

[5] Ottava, Prager medicinische Wochenschrift, xiv., 1893.

[6] Von Graefe, Archiv für Ophthalmologie, iii. 1.

[7] Von Hasner, Beiträge zur Physiologie und Pathologie des Auges, Prague, 1873, S. 57.

[8] Gruening, On the Operative Treatment of Divergent Squint, New York Medical Journal, No. 693, 1892, March 12.

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(*B*) ADVANCEMENT OF THE MUSCLE; PRORRHAPHY.

(1) A. von Graefe improved the attempts of J. Guérin into a definite method, which, though now obsolete, deserves to be mentioned. In divergent squint he detached the conjunctiva from the corneal margin and undermined it back to the caruncle. After that he severed the retracted muscle from the sclerotic, incised the conjunctiva over the tendon of the antagonist, passed a suture through the scleral end of the tendon, cut the tendon behind it, and drew with the thread the eye so far to the other side that the edge of the loosened, formerly retracted, tendon of the internus reached and even overlapped the border of the cornea. The eye was kept in this position by fastening the thread over the nose with strips of adhesive plaster.

This method, called the thread operation (*Fadenoperation*), frequently yielded good results, but was cumbersome and not without danger, suppuration of the cornea having followed it. Von Graefe himself, therefore, gave it up very soon in favor of

(2) *George Critchett's method of advancement by stitching the tendon forward.* This operation, variously modified, has been generally adopted ever since Critchett communicated it at the Heidelberg Congress in 1862. I have used it in the following way. Supposing, for example, that the internal rectus was to be advanced in secondary divergent strabismus. Under general anæsthesia, sometimes under local or no anæsthesia, I first make a free tenotomy of the external rectus. Then I incise the conjunctiva on the nasal side vertically about ten millimetres, four or five millimetres behind the margin of the cornea. I undermine the conjunctiva from the incision medially as far as the cornea, and obliquely above and below around the cornea as far as the vertical meridian. Now I undermine the conjunctiva two or three millimetres downward towards the caruncle, divide Tenon's capsule at the lower border of the tendon, pass the squint-hook under the tendon, dividing successively all the adhesions of the tendon and muscle that I can discover; at the same time I divide Tenon's capsule above and below the tendon. During all this probing and cutting the hook remains under the conjunctiva until no more adhesions are felt. When it is raised it is loaded with conjunctiva, tendon, muscle, and connective tissue, all of which I seize with ordinary, broad, fixing forceps, applying one prong to the inner, the other to the outer (conjunctival) surface. I now withdraw the hook and apply the sutures, never less than three, mostly four, in number. The needles are very sharp, not too much curved, slightly flattened, so as to pass with as little pushing as possible through the outer layers of the sclerotic. The thread is thoroughly sterilized, strong, and not too thin, so that it neither loosens in tying nor cuts through the advanced muscle. The two peripheral needles, which are the most effective, are passed most posteriorly through the flap, in high degrees of divergence directly before the semilunar fold, the middle sutures

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somewhat more in front. One of the middle sutures is applied the first, then the lateral ones, and lastly, if thought needful, the other middle one. All the needles are firmly grasped with a Sands' holder and first thrust perpendicularly through the whole thickness of the flap, then the middle sutures are thrust under the conjunctiva through the outer layer of the sclera as near to the corneal margin as practicable. After that the lateral sutures are passed three or four millimetres obliquely under the bulbar conjunctiva and pushed boldly through scleral tissue, so as not to cut through now or later. They emerge, if a great effect is required, close by the vertical meridian. When all the sutures are in, a portion of the tendon is cut off. During the cutting and the introduction of the suture the wound and cornea have to be kept moist with a mild antiseptic. While the sutures are being tied, an assistant holds the eye turned towards the nose with a forceps applied to the neighborhood of the lateral wall of the cornea. Many operators advise that the lateral sutures be tied simultaneously, one by the operator, the other by an assistant, in order to avoid a deviation up or down by unequal traction of the sutures. The advice seems good, but I must say that I never followed it, and have not met with a secondary deviation. During the application of the sutures we must watch their effect, and graduate the tightness of the ligatures accordingly. The threads ought not to be cut too short, otherwise they cannot readily be taken up when removing.

There should be a convergence of about four millimetres, but no more. If this is not obtained when all the sutures are tied, I examine the external rectus for an irregular insertion of some part of the tendon. In case I do not find any, I cut the check-ligaments more freely; and if this also fails to give the desired convergence, I may, as I have sometimes done, "hitch" the eye to the inner commissure in the manner described above, passing the needle through one of the middle sutures. The eye is cleansed and bandaged. The patient has to keep his bed two or three days, and his room a week. The sutures are removed according to the effect of the operation. If the convergence has not diminished on the fourth day, I remove one or both middle sutures. Otherwise, I leave them in six days. The wound then commonly begins to swell, for the tightness of the sutures may set up inflammation, even if the silk was absolutely aseptic when used.

(3) *Schweigger's method of advancement: exsection of a piece of tendon, stitching the two edges together.* Schweigger begins with the tenotomy of the antagonist, then divides vertically, about ten millimetres in extent, the conjunctiva over the insertion line of the tendon to be advanced, lays the tendon and muscle bare, frees their borders from Tenon's capsule, passes a hook under the anterior end of the tendon and another more or less farther back, according to the degree of the deviation. He then measures off with a small millimetre ruler how much of the tendon he wants to exsect,—namely, a piece about as large as the degree of the deviation. To make this measurement he applies the little ruler to the tendon, which is gently stretched between the two hooks. After this determination he passes one doubly

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armed catgut suture through the upper and another through the lower half of the muscle, immediately before the posterior hook. He first passes one needle from above under the muscle, and pierces the latter from the inner side outward, a little below the middle. The second needle is passed from below and inward to a little above the middle of the muscle. He now ties both sutures so that the whole muscle is ligated, cuts the muscle immediately in front of the sutures, and the tendon near its insertion, leaving a small stump through which the needles of the catgut threads are passed. With these threads the muscle is drawn forward and tied to the stump of the tendon. The conjunctiva is stitched over the wound with fine silk.

By this operation the eyeball is mostly turned as much as the exsected piece of tendon and muscle is long.

VARIETIES OF THE ADVANCEMENT OPERATION.

The numerous modifications of the advancement operation which have been devised and published rest on the original prorrhaphy of Critchett. They differ in the number of sutures and in the way of applying them. I can mention only a few of them.

(1) The *pulley operation* of A. E. Prince[1] has been favorably received. It was modified in its turn by Berry.[2] The sutures are supported by a vertical thread. The needles of a doubly armed thread are introduced from the inner side of the detached tendon, one at the upper, the other at the lower border, through muscle, Tenon's capsule, and conjunctiva. One needle is then drawn through the loop and the two threads are tied. In this way the muscle is moved forward as a weight by a pulley.

Instead of two sutures, one suture will advance the muscle in a similar way. A thread armed with two needles is introduced from the scleral side of the detached tendon, as in the pulley operation. Either needle is then passed vertically through the conjunctival and episcleral tissue two millimetres from the corneal margin, and the muscle drawn forward by tying the two ends of the threads in a horizontal direction.

Valude splits the end of the detached tendon in the middle, and stitches one half up, the other down to the sclera, near the vertical meridian. In this way he secures a great and symmetrical bringing forward of the tendon.

H. D. Noyes's[3] modification of Critchett's prorrhaphy deserves a more extensive review, because it illustrates some of the most important points. The author says (p. 173), "I find no need of the hitching thread of Agnew, nor the clamp forceps of Wecker, nor the anchoring thread of Prince. The ordinary fixation forceps, having a spring-catch, gives perfect control of the muscle." He applies three sutures of fine, black, strong silk. "The im-

[1] A. E. Prince, Ophthalmic Review, 1887, and Archives of Ophthalmology, vol. xxii. 1893, p. 498.

[2] G. A. Berry, Diseases of the Eye, second edition, 1893, p. 702.

[3] H. D. Noyes, A Text-Book on Diseases of the Eye, second edition, 1894, p. 172.

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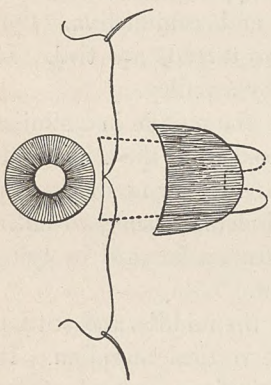
portant thing is to have the needles . . . ground so sharp that they could as easily as a discission-needle penetrate the cornea. On their fineness, sharpness, temper, and curve [so-called half-curve, see Fig. 38] success chiefly depends." He grasps the tendon with a fixation forceps, detaches it, shuts the spring-catch, raises the tendon, and introduces one needle of a double-armed thread from the sclerotic outward through the middle of the capsule, muscle, and conjunctiva, cuts away superfluous tissue in front of the needle, and draws the needle through. In the same manner he applies a suture through the upper and another through the lower part of the muscle. He forms a conjunctival flap six millimetres in width from the insertion of the tendon to the cornea. Then he passes the needles at the other ends of the threads beneath the conjunctiva through the outer layers of the sclerotic, so that their points emerge at the limbus of the cornea. "To get them through without breaking, they must be seized at the middle and pushed without any lever action. If the globe tends to rotate, it may be steadied by a bident. What is essential is to have a firm hold on the sclera."

Excision of pieces of the tendon and muscle have been made and described by J. F. Noyes,[1] of Detroit, Vieusse,[2] Driver, Coates, Baraquez, Prince, Stevens, Müller, and others.

FIG. 40.



Advancement operation by folding the muscle according to Dr. Lagleize, of Buenos Ayres. (After Panas.)

Shortening of the muscle by folding was done by (1) J. F. Noyes, of Detroit.[3] The tendon is divided near its insertion, and the distal end brought forward under the proximal end, lapped, and sutured. At the same time a portion of the tendon may or may not be cut off. The point of insertion of the tendon is not changed in this method. The folding of a piece has, of course, the same effect as if so much of the tendon had been excised.

(2) Lagleize,[4] of Buenos Ayres, makes a neat folding operation as follows. He excises a piece of conjunctiva over the tendon (Fig. 40), passes a hook under the tendon near its insertion, and another under the muscle. He passes one of the needles of a doubly armed thread near the posterior hook, one millimetre from its border, from above and inward, through the muscle and conjunctiva outward, and the other needle the same way from below. He then carries the needles under the con-

[1] J. F. Noyes, A New Method of Operating for Strabismus, Transactions of the American Ophthalmological Society, 1874, p. 273.

[2] Vieusse, Du traitement chirurgical du strabisme (nouveau procédé opératoire), Recueil d'Ophtalmologie, 1875, p. 330.

[3] Loco citato.

[4] Lagleize, Archives d'Ophtalmologie, t. xii., 1892, p. 668.

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junctival bridge to the cornea. By pulling at the threads the muscle is drawn forward over the tendon, after which the threads are united near the cornea. The more strongly one pulls the more the deviation will disappear. He removes the sutures in twelve days.

The author supports the claims of efficiency for his method by three successful cases where the deviation had varied between twelve and fifty degrees.

The operation is simple, and seems to admit of a nice graduation of the effect, at least if the threads are passed through the outer layers of the sclerotic so as not to give way.

Grandclément[1] operates in cases of inveterate divergent strabismus, if tenotomy of the external rectus is not sufficient, as follows. With a pair of fixing forceps he raises, near the internal canthus, a vertical fold. The needles of a doubly armed suture are passed horizontally so deeply through the tissue that not only conjunctiva but also capsule and tendon are comprehended in the loop which is formed by the tying of the thread. The thread remains *in situ* two or three weeks without causing discomfort.

OPERATIONS ON TENON'S CAPSULE.

The knowledge of the anatomy of the ocular aponeurosis which envelops all the muscles, to subserve their movements as a synovial membrane and steady and protect the eyeball as suspensory and inhibitory ligaments, is of great importance in the understanding and treatment of the anomalies of motility, as we have seen in many places of this section. Of late *operations on this aponeurosis alone* have been described by which the movement of the eye can be increased, as in advancement, or diminished, as in tenotomy. Of each kind only one operation has come to my knowledge,—namely, the capsular advancement by de Wecker and the capsulotomy by Parinaud. I shall describe the latter first, though it was invented later.

(1) *Division (setting back) of Tenon's capsule by Parinaud:*[2] *diminishing the action of the muscle by cutting its check-ligaments* (recul des ailerons latéraux). In a case of convergent strabismus the eye is drawn forcibly towards the temple by an assistant, and a conjunctival fold, raised with forceps, is cut vertically to the extent of from ten to fifteen millimetres, about seven millimetres behind the corneal margin. The conjunctiva is undermined over the tendon and muscle back to the caruncle; the tendon is exposed and the capsule cut along its edges. Through these apertures a pair of curved scissors is introduced and two incisions are made of from eight

[1] Grandclément, La cure du Strabisme obtenue ou complétée par un simple froncement souconjonctival du muscle et de la capsule, à l'aide d'une anse de fil, Bulletins et Mémoires de la Société française d'Ophtalmologie, 1893, p. 291.

[2] Parinaud, Opération du strabisme sans ténotomie, Note à l'Académie des Sciences, 14 Avril, 1890, and Rapport sur le traitement du strabisme, Bulletins et Mémoires de la Société française d'Ophtalmologie, 1893, p. 137.

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to ten millimetres, one upward, curving a little backward, the other downward, also curving backward.

After this a capsular advancement is made on the antagonist, and if the effect is still insufficient, Parinaud divides the tendon too. The wound is closed with conjunctival sutures, which are removed in one or two days; the sutures in the capsule of the antagonist in five or six days.

(2) *Capsular advancement by de Wecker.*[1] Wecker at first excised an elliptical piece of conjunctiva over the tendon, but now he also makes only a vertical incision, undermines the conjunctiva towards and around the cornea as far as the vertical meridian, opens Tenon's capsule at the lower border of the tendon, passes a hook below the tendon, incises upon it the capsule on the upper side of the tendon, then passes a suture through conjunctiva and episclera from a point near the lower portion of the vertical meridian, coming out in the conjunctival defect, then introduces it into the opening in the capsule, advances it along the edge of the muscle, and pushes it out through the tissue of the capsule and the conjunctiva, the farther back the greater he desires the effect to be. Another suture is passed in the same way through the episclera and capsule above the tendon. The threads are united and the eye bandaged.

I saw this operation performed by de Wecker in January, 1886, and after my return to New York made the operation a few times. The effect proving insufficient, I at once modified it in such a way that it was in reality a

(3) *Tendino-capsular advancement*, and performed as follows.[2] The conjunctiva is vertically incised over the insertion of the tendon, the conjunctiva undermined around the cornea to the vertical meridian, Tenon's capsule opened with scissors at the lower end of the insertion line of the tendon, a strabismus-hook slipped underneath the tendon, and the capsule incised over the tip of the hook on the upper side of the tendon. Three or four sutures are applied, one through the conjunctiva and lower edge of the muscle, passing under the conjunctiva obliquely forward, then two or three millimetres through the outer layer of the sclera, immediately before emerging on the conjunctiva near the vertical meridian. The second suture is on the upper side, and pursues a course analogous to the first. The third suture is passed through the conjunctiva and the middle of the muscle, advanced *under* the hook, which during the application of all the sutures raises the tendon, thrust through the middle of the tendon near its insertion, then through the superficial layers of the sclerotic, to emerge on the conjunctiva near the cornea. If a large effect is desired, a fourth suture

[1] De Wecker, Sur l'opération du strabisme au moyen de l'avancement capsulaire, Annales d'oculistique, t. xc., 1883, p. 188; La combinaison de l'avancement capsulaire et de la ténotomie, Annales d'oculistique, t. xciii., 1885, p. 72; Les opérations modernes du strabisme, Archives d'Ophtalmologie, t. xiii., 1893, p. 1.

[2] H. Knapp, Advancement of Tenon's Capsule, Transactions of the American Ophthalmological Society, July, 1886.

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is applied at the side of and similar to the third. The sutures are tied in the same way as in Critchett's advancement, described above.

The operation, in fact, is the same as Critchett's without excision of a piece of the tendon. The sutures remain five or six days. The operation acts by shortening (tightening) the check-ligaments and the muscle *by folding them.* They remain folded and attached to their new position on the sclerotic by means of cicatricial tissue, which is formed by an adhesive inflammation due to the irritation set up by the sutures. I have had an opportunity to convince myself of this fact by a secondary tenotomy of an internal rectus after an over-effect of the above tendino-capsular advancement in a case of primary divergent strabismus.

PARTIAL TENOTOMIES AND PARTIAL ADVANCEMENTS.

Partial tenotomies have been made by many operators. Von Graefe tried them. I did it myself at the beginning of my practice, and when, in conversation, I told von Graefe of it, he answered, "You will not do that long."

Arlt[1] says, "If I made a tenotomy for simple strabismus, or for insufficiency, and, afraid of an over-correction, left only the one or the other fibre undivided, the effect always was only transitory."

Recently, on the recommendation of George T. Stevens,[2] they have been extensively practised in the United States of America. A button-hole in the centre of the tendon is extended by successive subconjunctival partial detachments of the tendon from the sclerotic up and down, and the effect measured after each cutting until it appears to be correct. The patient is examined each day, and when the effect is insufficient, another portion of the tendon is cut, and so on until the desired effect proves permanent. Should, after the first or any subsequent operation, the effect be found excessive, it is reduced by restrictive sutures. These are the long-known procedures to graduate the effect of tenotomies, but it is difficult to determine how much, if not the whole, of the tendon had been divided before the effect became adequate.

Partial advancements are also recommended and done. Either central or peripheral portions of the tendon have been excised and the shortened portions stitched forward. I have not seen any lasting effect from the small triangular excisions and *prorrhaphy* of the central portion recommended by Stevens,[3] but have noticed decided effects from division of the peripheral parts, leaving a small central portion intact. The effect was proportionate to the amount of tendino-capsular advancement by which the preserved central portion was folded.

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[1] Operationslehre, in Graefe-Saemisch, Handbuch der gesammten Augenheilkunde, Bd. iii. S. 402.

[2] Archives of Ophthalmology, vol. xvi. p. 149, vol. xvii. p. 155, vol. xviii. p. 371, vol. xx. p. 356, and Archiv für Augenheilkunde, Bd. xviii. S. 445, Bd. xxi. S. 325.

[3] Archives of Ophthalmology, vol. xviii., 1889, p. 384.

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GENERAL REMARKS ON THE ADVANCEMENT OPERATIONS.

(1) *As to their Safety.*—The operations for advancement are so extensive in comparison with the smallness of the organ on which they are performed, that naturally we might have some doubt as to their safety. Yet experience has shown that, with the exception of Graefe's thread-operation, they are surprisingly harmless. I have made many advancements, and do not remember a single case in which alarming symptoms have appeared, nor has such a case come to my notice from literature or other operators. I do not doubt that cases of severe, perhaps disastrous, reaction have occurred, and am perfectly prepared to believe that such a case may any day present itself to me. Yet I no longer undertake these operations with such misgivings as I formerly had.

In some cases there is *a certain disfigurement*—namely, a permanent thickening of the conjunctiva at the place of the wound—left behind. Some methods are more apt to produce it than others. The worst is the tendino-capsular advancement, and even in these cases it is not lasting. It looks bad enough in the first weeks, but diminishes from month to month, so that the redness disappears in three months and the swelling in about six, being scarcely noticeable later.

The *semilunar fold and caruncle are sometimes drawn towards the cornea*, but not in a particularly disfiguring degree. Never have I seen or heard that pterygium followed an advancement operation.

As to the reaction, there is some pain in certain cases during the first days. The swelling of the conjunctiva is sometimes considerable, especially on the sixth or seventh day, but it disappears soon after the removal of the sutures. Never have I seen purulent secretion or corneal ulcers follow an advancement.

(2) *As to the effect* of these operations I can say that it varies greatly according to the method used, and in any one method according to the way in which the operation is done. With many of the methods smaller or larger effects can be obtained at the will of the operator, as I have stated in describing the operations of Critchett and Schweigger and the tendino-capsular advancement.

Other operations, especially those where only one doubly armed thread is used, seem less reliable when larger effects are required. This is, however, so much a matter of personal experience that I am loath to pass judgment on procedures I have not tried. I can say that with the Critchett four-suture advancement any effect to satisfy the greatest demands is obtainable.

The *effects of advancement operations* in the same hands with the same method *vary* considerably *with the conditions of the case.* Thus it is well known that secondary divergent strabismus yields better results when operated upon than does primary divergence. The weaker in itself the muscle is the less will be the result. We can with less trouble and a high

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degree of accuracy correct the worst cases of secondary divergence, whereas in paralytic strabismus the permanent gain is often insignificant.

INDICATIONS AND RESULTS OF SQUINT OPERATIONS.

(1) In *comitant strabismus*, the *single simple tenotomy*, strictly limited to the tendon of one eye, is the operation for *low degrees of convergence*, be the strabismus constant or alternating. Under the precautions detailed before (see p. 875), the operation is perfectly safe and should not leave any unpleasant consequence.

In *medium and higher degrees*, the *double simple tenotomy—i.e.*, the unextended division of the tendon of each internal rectus—is indicated, and will produce just as perfect results as the single tenotomy in the low degrees.

In support of this statement I can demonstrate to-day numerous cases, operated on from fifteen to twenty-seven years ago. They have, if not binocular vision for all tests, at least binocular fixation, and so natural a position and such free movements of the eyes that nobody, even on functional examination, would find out that there ever had been a squint. In this way the movements and positions of both eyes are equalized; and the method is all the more to be recommended since in medium and higher degrees of convergence the area of motion is shifted towards the nose in both eyes. This group of squint cases is perhaps more numerous than all other varieties together.

Of late the advantages of advancement have been so much and so justly dwelt upon by Landolt, Wecker, and others that this operation has now come into greater favor than, in my opinion, it deserves. During the last twelve years I have made many advancements in comitant convergent strabismus, always with tenotomy of the antagonist, yet gradually I have returned, in the majority of cases, to the double tenotomy.

In *divergent strabismus* a single, even most extensive, tenotomy is hardly ever, that of a double tenotomy rarely, sufficient to produce a permanent recovery. Here advancement of the antagonist has to supplement the tenotomy of the contracted muscle. This is principally the case when the eye is prominent and the adduction weak. *Free tenotomy of the external rectus combined with advancement, best a tendino-capsular advancement, of the antagonist*, is suited for these cases, and if the effect is not sufficient, tenotomy of the external rectus of the other eye, likewise combined, if needful, with advancement of the antagonist, will scarcely ever fail to produce a good result.

In cases of *inveterate unilateral convergent strabismus*, in which the *fixing eye* has not only *good vision* but also tolerably good abduction, whereas the *other eye* is *very amblyopic* and half hidden behind the caruncle, with marked diminution of abduction, we may get along with *tenotomy of the internal and advancement of the external rectus on the squinting eye alone;* and we cannot do otherwise, if the patient refuses to have the "good eye meddled

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with." In such cases combination of tenotomy with advancement of the antagonist is vastly superior to tenotomy alone. In order to get a sufficient effect by tenotomy alone, we must make it very extensive. This will not simply turn the eye outward, but by weakening the check-ligaments will also move it forward until the diminished force of the retracted adductor counterbalances the weak and stretched abductor. In the childhood of the squint operation, when only the deviated eye was attacked, the insufficiency of the primary effect in high degrees of convergence led the surgeon not only to cut as much as he could, but to shift the tendon and its surroundings back towards the equator with a kind of blunt hand-chisel, which the late Professor Böhm, in Berlin, showed to the students as the instrument that did so much mischief and produced those horrible degrees of exophthalmic divergent strabismus which for a time made many ophthalmic surgeons give up operating for squint altogether. *Advancement supplements tenotomy in such cases admirably: by shortening the antagonist it increases its power, keeps the eyeball back,* and *makes an extensive* (*weakening*) *tenotomy unnecessary.*

In *insufficiency* (heterophoria) we must carefully determine its *degree* and the *power* of the muscles (abduction, adduction, sursumduction, and deorsumduction). We know that in the normal state abduction is seven degrees, sursumduction or deorsumduction two or three degrees, adduction so variable that from about twelve degrees at the first examination it can by further trial soon be brought to thirty and forty degrees. We must ascertain also the near point of binocular fixation (F. p. = fusionis punctum proximum), which, together with the abduction, determines the range or amplitude of convergence.

Before we decide to operate on a case of heterophoria we must ascertain by prolonged trial how much benefit the patient derives from spherical, cylindrical, and prismatic glasses. If spheres and cylinders do not help him, but prisms do, he can be operated on with a fair chance of success. Yet, if weak prisms (three degrees or less before each eye) make him comfortable, we may abstain from operating; but if only stronger prisms give relief, I consider an appropriate surgical interference with the muscles to be preferable.

The procedure should be *tenotomy* according to Snellen's (button-hole) method, *with careful graduation* of the effect as described before (page 863), capsular or better tendino-capsular advancement, also with careful determination of the effect. Advancement operations alone—*i.e.*, without tenotomy of the antagonist—may be efficient when only small errors of motility have to be corrected, as in esophoria and hyperphoria, but in exophoria they scarcely ever will answer; on the contrary, in many cases, especially when the eyes are prominent, a permanent correction will require tenotomy of the external rectus and advancement of the internal in both eyes.

The *results* of the *insufficiency operations* are gratifying in a consider-

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able number of cases, in others, perhaps in an equal or greater number, they are disappointing. The heterophoria, lateral, vertical, or both, is perfectly corrected, the patient feels relieved for months, then his headache and asthenopia return, while the muscular dynamics, examined with all the means at our command, remain normal.

The distressing symptom of *cephalalgic asthenopia*, which drives so many—mostly young—people to the oculist, should be carefully examined as to its local and general causes.

Not only patients with asthenopic but also many with *pure headache*, which the family physician fails to relieve, are now sent to the specialist. It is the duty of the latter, after a thorough physical and functional examination, to confer with the family physician, but under no condition should he be induced to try an operation if he finds no well-defined muscular abnormity the correction of which would in itself promise an improvement in the dynamics of the eye. Not a few patients have come to me complaining that their muscles had been cut three, four, or more times. Their headache, they said, was as bad as ever, and whereas they could use their eyes before, they could not do so now.

The same rule holds good for the *pure ocular* or *ciliary neuralgia*.

The operative treatment of *paralytic affections* of the eye requires great caution as to their indications. If a person has a paralytic squint with diplopia, the operation will mostly do him more harm than good. The disfigurement may be lessened, but the diplopia will be more distressing on account of the approximation of the two images.

When we operate solely for cosmetic purposes, one eye being blind, our efforts will be baffled, for if we advance the paralytic muscle with or without weakening the antagonist, the eye will ultimately return to its previous position. When a perfectly powerless muscle is opposed by another with never so little power, the continuance of the action of that power will draw the eye more and more to the side, and the effect of this action will stop only when there are insuperable mechanical obstacles in the way.

If the paralysis, though chronic, is *amenable* to *treatment*, for instance in syphilitic disease, or is *spontaneously improving*, that improvement may be favorably influenced by a tenotomy, for if the resistance is diminished the action of the paretic muscle will have a more telling effect on the motility of the eye. The paretic muscle will be less exhausted by its work, and will gain strength by the exercise which it is now capable of taking. I have had several very satisfactory cases of that kind in my own practice.

Even when the *paresis is permanent* (*paretic strabismus*), an operation for relief of the diplopia may be undertaken with more or less success. Suppose a former paresis of the superior oblique of the right eye left a hyperesophoria or a simple hyperphoria, we have *three ways of* surgical interference before us.

(1) The *tenotomy of the chiefly contracted muscle*,—*i.e.*, the superior rectus.

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(2) The *advancement of the antagonist,—i.e.*, the inferior rectus of the same eye.

(3) The *tenotomy of the antagonist of the other,—i.e.*, the inferior rectus of the left eye.

It will depend on the relation of the muscular forces of the two eyes, also on the occupation of the patient, which of the three methods in a given case is likely to yield the best results. I have favored the first method, as the simplest,[1] and have had satisfactory results. Alfred Graefe criticises this method, and, like his great relative A. von Graefe, he prefers in the majority of cases the tenotomy of the antagonist of the other eye. In the latter case the deorsumduction would be weakened, but equal in both eyes. I am loath to weaken a muscle because its partner is weak. Von Graefe even went so far as to recommend in a case of paralysis of one lateral muscle the tenotomy of the three others in order to equalize the muscular forces. It appeared to me a very difficult problem to produce equilibrium in four muscles by tenotomizing three strong muscles to harmonize their action with a paretic fourth. In the last thirty years operations for insufficiency have been rather popular in America. They consisted chiefly in tenotomies of the stronger muscle, or advancement of the weaker, or both, whereas tenotomy of the antagonist of the other eye has been rarely resorted to. This agrees exactly with my views. A careful analysis will show which of the three ways, or what combination of them, is most likely to give satisfactory results.

In conclusion, let me say a few words about the *proper time to perform muscle operations.* In exceptional cases we may, in my opinion, operate on *young children,* even during the first year of life. I have done this in high degrees of congenital convergent strabismus. The examination must exclude paralysis and cerebral disease still present. If in higher degrees of comitant strabismus the physical and functional examination as far as feasible prove otherwise normal, a careful tenotomy on the eye most deflected will place the child in favorable conditions for obtaining in its natural development straight eyes and binocular vision, whereas without an operation the excessive convergence might be an insurmountable obstacle.

In the ordinary comitant strabismus beginning in the second or third year, or later, the optical treatment should be used, and in addition the deviated eye should be strengthened by frequent covering of the other. It is now generally believed that an operation should not be made before the seventh year. This has been my practice also, except in cases of excessive strabismus. If glasses had been constantly worn for one or several years without showing a decided improvement, I have operated on one eye earlier than in the seventh year, and found that then the optical treatment was of more benefit.

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[1] See H. Knapp, Three Cases of Tenotomy of the Superior and Inferior Recti, with Incident Remarks, Archives of Ophthalmology and Otology, vol. iv., 1874, p. 20, and Archiv für Augenheilkunde und Ohrenheilkunde, Bd. iv., 1, S. 92.

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Both eyes should not be operated on at the same time, unless there be a special reason for doing so. It is better to delay the second operation until we can approximately judge what the definite effect of the first operation will be. If there is still enough convergence and, as usual, also contracture of the internal rectus of the other eye, this may then be operated on in five or six days, or later. The advantage of equal muscular action in both eyes is a sufficient reason for not delaying the second operation very long. When, however, after the first operation the movements of the eye have become irregular, and the eyes, with or without glasses, at times are more or less straight, or show a tendency towards divergence, then again converge greatly, we should put off the second operation until the condition of the eyes has assumed a definite character.

There is no harm done in delaying the squint operation until the age of puberty or one or several years later. The surgeon then has the advantage of being able to make a more thorough examination, and of having a more intelligent and docile patient to deal with, making it easier to perform the operation and to determine its primary effect and conduct the after-treatment. I think the earlier school years are the most suitable time. The operation can then be done with sufficient ease and accuracy, the child is spared a good deal of vexation on the part of the other children, and is in better condition for developing binocular fixation, especially if the operation be supplemented by glasses. The optical treatment alone, especially with a view to raise the acuteness of vision, the so-called *orthoptic treatment*, which Dr. Javal, of Paris, has so persistently studied, and in its nature and possible results so exhaustively described,[1] is not to be relied on when the strabismus is constant.

§ VIII. OPERATIONS ON THE EYEBALL.

There are three operations on the eyeball that deserve to be described in particular,—enucleation, evisceration, and evisceration followed by insertion of a glass bead into the vitreous chamber. The first is the most important, the last the least.

I. ENUCLEATION, SHELLING OUT OF THE EYEBALL.

The ball is removed with preservation of the conjunctiva, muscles, and all the other contents of the orbit. It was first done by Bartisch, of Königsbrück (Saxony),[2] rather rudely with a sharp spoon, which he passed behind the eyeball from all sides. The true enucleation was first described[3] and recommended by Bonnet, of Lyons, in 1841, and executed by Stoeber, of Strassburg, in 1842. "The chief merit of Bonnet's operation, according to

[1] E. Javal, Manuel du strabisme, 1896.

[2] Bartisch, Ophthalmodouleia oder Augendienst, Dresden, 1583. A quaint old folio, with many crude illustrations; not very rare.

[3] Bonnet, Traité des sections tendineuses et musculaires, Lyon et Paris, 1841, and Annales d'oculistique, t. v., 1841, p. 27; t. vii., 1842, p. 39.

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Panas (*Traité*, t. i. p. 379), consists in the conservation of Tenon's capsule, so that the eyeball can be removed without injuring the soft parts of the orbit." With this I fully agree. It was modified by several surgeons. Arlt's modification only need be described.

No new instruments are required, except, perhaps, a pair of curved scissors, a little stronger than squint scissors, for cutting the optic nerve. General anæsthesia is advisable, yet the operation is neither so painful nor so long as to necessitate more than local anæsthesia. Injection of a two per cent. cocaine solution behind the eye is sometimes followed by unpleasant symptoms, excessive pallor, cold perspiration, and syncope. Dropping cocaine into the wound as the operation proceeds is tedious and not perfectly efficient.

(1) *Bonnet's method*, the most popular, is as follows. The conjunctiva is incised with strabismus scissors all around near the corneal margin, and dissected from the sclerotic as far as the insertions of the tendons of the straight muscles. The tendons are severed as in squint operations, one after the other, together with the subconjunctival connective tissue, close to the sclerotic and backward beyond the equator of the globe. Now the eyeball can be dislocated forward by pressing the wire speculum back. This facilitates the division of the optic nerve. If the eyeball does not readily protrude, the optic nerve can almost as easily be cut as if the globe were dislocated. In both cases the eye should preserve its natural straightforward position without rotation on its antero-posterior axis. A pair of curved scissors, somewhat stronger than squint scissors, are held closed and pushed backward between the denuded sclerotic and the detached conjunctiva until the hard and tight cord of the optic nerve is felt. Having determined the exact position of the optic nerve by moving the end of the scissors up and down, we slightly withdraw the scissors, open their branches, get the nerve between them, and cut it close to the sclerotic. The globe now protrudes readily, and is held with the fingers of the left hand until the insertions of the tendons of the oblique muscles, the ciliary vessels and nerves, and the delicate connective-tissue fibres are severed from the sclerotic.

In all this cutting it is of paramount importance to *avoid injuring Tenon's capsule* and cutting into the cellular tissue of the orbit. As the orbital tissue is very vascular, in some cases almost erectile, wounding it may be followed by inordinate hemorrhage, or by orbital cellulitis, which not very rarely terminates fatally. This caution is imperative if we have to operate under septic conditions, such as panophthalmitis.

In cutting we should always *turn the points of the scissors towards the eyeball*, not away from it; the orbital tissue has to be spared; the eyeball is the foreign body, the tumor that has to come out. I do not mean, however, that we could permit ourselves to manage the eyeball inconsiderately; on the contrary, we should be most careful not to cut into it, for this would make it collapse and flood the wound with its contents. If we shell the eye strictly out of Tenon's capsule without cutting into the orbital tissue,

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we protect the latter from infection, secure the smoothest recovery, and obtain the best support for an artificial eye.

(2) *Arlt's method*[1] is a modification of Bonnet's. As in his squint operation, Arlt does not use a hook. He incises the conjunctiva from two to three millimetres behind the limbus corneæ and pushes it somewhat back. Standing on the right side of his patient, he in the left eye divides first the external, in the right the internal rectus, grasping it with toothed forceps, but leaves a small stump to get a firm hold of the globe with the forceps. After division of the inferior and superior recti, he pulls the eye with the forceps horizontally towards the inner (or outer) canthus, passes a pair of scissors over the posterior segment of the sclerotic as far as the optic nerve, opens the branches, advances the scissors so that the nerve lies between the branches, and cuts it close to the sclerotic. He now turns the protruding eyeball to the side of the uncut rectus, divides the insertions of the obliques and the vessels and nerves at the posterior half of the globe, and lastly detaches the insertion of the fourth rectus, together with the overlying conjunctiva, from the sclerotic.

I have no experience with Arlt's method, but think it is very good. Czermak,[2] a pupil of Arlt, calls it "classical, unsurpassed by any of the different variations,—a model of simplicity and perfection in detail." I have practised Bonnet's method in the following manner: The conjunctiva is incised near the nasal corneal border, detached up and down around the cornea about as far as the vertical meridian, the inner rectus divided on the hook from below upward, the subconjunctival tissue detached on the hook above the upper border of the internal rectus; the hook is passed under the tendon of the superior rectus, which, together with conjunctiva in front of the insertion of the tendon, is detached from the sclerotic in strokes passing obliquely from the nasal to the temporal side, according to the curved insertion of the tendon, from in and down to out and backward;[3] then the hook is passed farther temporally under the conjunctiva, which is detached from the sclerotic as the hook advances towards the external rectus. The hook is now withdrawn and inserted under the tendon of the inferior rectus, which is cut, together with the conjunctiva, over and before it, also obliquely from above and in to out and slightly backward. The hook is then advanced temporally under the conjunctiva and the insertion of the external rectus, which are divided as the hook advances until it reaches the point where it was withdrawn after the tenotomy of the superior rectus. To be sure that the whole conjunctiva and all the tendon fibres are detached, the hook is passed through the whole wound as far back as the equator, and any fibres that have escaped are severed with the scissors. The eyeball is now raised with the point of the scissors, the optic nerve, the insertions of

[1] Arlt, Zeitschrift der Wiener Aerzte, 1859; Operationslehre, in Graefe-Saemisch Handbuch der gesammten Augenheilkunde, Bd. iii., 1874, S. 415.

[2] Czermak, Die augenärztlichen Operationen, S. 407.

[3] See Fig. 31, in anatomical introduction to § VII. (Operations on the Muscles).

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the obliques, and the remaining tissue cut close to the sclerotic, and the eyeball removed. When the bleeding is stopped, the conjunctival wound is closed by a "bag-mouth" suture,—viz., with a curved needle a thread is passed through the edge of the conjunctiva at intervals of from three to five millimetres, then tied like the mouth of a bag by a string. The eye is closed with the ordinary gauze-cotton bandage.

COMPARISON OF THESE METHODS.

I think it preferable to *begin in each eye with the tenotomy of the internal rectus*, for it is easier and surer to tenotomize the vertical muscles from the nasal towards the temporal side, on account of the temporal ends of their insertion curving irregularly backward.

In *dividing the conjunctiva successively as the hook advances* from tendon to tendon, we can preserve more conjunctiva than if we divide the conjunctiva all around the cornea before we cut any tendon, because we cannot pass the scissors under the conjunctiva nearer than about three millimetres from the corneal margin. It is important to preserve all connections of mucous and fibrous tissue with the tendon, in order to prevent the retraction of the tendon as much as possible.

The *closure of the conjunctival wound by a suture favors primary union*, whereas when it is omitted a button of proud flesh not infrequently sprouts from the opening. Czermak [1] says, however, "I avoid all suturing of the conjunctiva, because it is unnecessary, does not accelerate the recovery, and always occasions a greater diminution of the stump." If I were prepared to take his word for the latter *ex cathedra* statement, I would at once give the suture up.

ACCIDENTS AND MISTAKES DURING THE OPERATION.

(1) *Hemorrhage*, sometimes copious and persistent, has occurred during the operation or later: one, ending fatally in spite of ligature of the common carotid, is on record. In most cases it is insignificant and easily stopped by a compressive bandage. Some operators put a piece of aseptic sponge or gauze on the wound behind the lids, leaving it on a few days. Others suture the wound, but leave a small part open to put a drainage-tube in. This, I think, is unnecessary.

Now and then the *eyeball is found too large to pass through the palpebral fissure.* In such a case we must satisfy ourselves that the optic nerve is really divided, and then enlarge the palpebral fissure by a simple cut through the outer commissure.

In the *presence of intra-ocular tumors we should be prepared to encounter extra-ocular extensions behind the visible part of the eye.* We should advance very cautiously, even explore the posterior episcleral region with a probe, and, if a tumor be found, remove it, together with the eyeball,

[1] Czermak, Die augenärztlichen Operationen, S. 418.

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without cutting into it. If we have reason to suppose, from protrusion of the eye, especially in gliomas, that the pseudoplasm has invaded the optic nerve, we may secure the latter by seizing it with curved ligature forceps three or four millimetres behind its entrance into the eye. The optic nerve is cut between the forceps and the eyeball; if sound, the forceps are withdrawn; if diseased, we can pull it out and cut it as far back as possible.

Perforation of the sclera may occur in soft eyes during the tenotomy or on cutting the optic nerve. If this accident takes place, we have to proceed deliberately in dissecting all tissues from the globe, as in spontaneous or traumatic perforations. The operation takes longer, but is neither difficult nor hazardous, and the recovery is regular.

In *purulent inflammations of the eye, panophthalmitis,* there is sometimes so much *thrombosis* in the orbital veins that the orbital tissue is altogether hard and *lardaceous,* and the eyeball has to be carved out of a stiff socket. If we take our time and have the sclerotic always before us, the operation can be correctly performed and the recovery is mostly undisturbed.

If one eye has an intra-ocular tumor or any other condition that does not show by ocular inspection, *we should be on our guard lest we take the good* eye out. This awful mistake is sensationally mentioned in text-books and periodicals: I do not know whether it has actually occurred, but the possibility is undeniable.

The *common course of recovery* is smooth. After the operation there is no pain, but sometimes slight œdematous swelling of the conjunctiva or suggillation of the lids with secretion. All this disappears in a week or sooner. In three or four weeks an artificial eye can be inserted, but it should not be worn the whole day during the first months.

In *rare cases enucleation is followed by orbital and palpebral abscess, thrombo-phlebitis,* and *fatal meningitis.* The general cause is infection of the wound from a septic focus, the eyeball itself, or the lacrymal sac. Von Graefe, after the observation of two fatal cases, *counteradvised the removal of panophthalmitic eyes.* Statistics show that among the causes of the fatal cases from enucleation panophthalmitis is represented by a disproportionately high number. On the other hand, there are fatal cases enough in which no septic condition of the eyeball existed, and, moreover, numerous cases of panophthalmitis have made a perfectly smooth recovery after enucleation, relieving the suffering of the patients at once. Many oculists nowadays consider panophthalmitis no contra-indication to enucleation. I myself have removed a number of panophthalmitic eyes without bad consequences in any, yet I perform the operation only for particular reasons, —for instance, great debility, advanced age, mental derangement, etc., of the patient, where a rapid and painless recovery is important; otherwise I let the suppuration take its course, relieving the suffering by free incisions of the sclerotic to diminish the painful increase of tension, especially in

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the period before the spontaneous perforation of the globe; also by poulticing and anodynes. It is alleged that panophthalmitis constitutes in itself a danger to life. This assertion is, it seems to me, not confirmed by general experience,—certainly not by my own. Exceptionally everything occurs, but I have never seen a fatal case of panophthalmitis that was free from a grave general or local complication. Apart from the greater risk the patient incurs by having a panophthalmitic eye removed than by leaving it alone, he is amply rewarded for the longer duration and greater painfulness of the recovery by obtaining a far better support for an artificial eye.

Orbital and *palpebral abscess*, to which enucleation in very rare cases gives rise, usually ends in recovery. *Thrombo-phlebitis* may be *simple* and *get well*, or it may be *infective, ending in recovery* under the picture of purulent orbital cellulitis, or *in death* by extension to the cavernous and other sinuses, causing meningitis.

VALUE AND INDICATIONS OF ENUCLEATION.

The loss of an eye is a misfortune to which no person should be subjected unless it is impossible otherwise to cure or avert a greater calamity, intolerable suffering, danger of life, or of blindness in both eyes. Enucleation is the mildest and safest operation to remove an eye. It leaves a half-empty orbit, sunken eyelids, lacrymation, and frequently mucous secretion. These symptoms, as well as the disfigurement, can be greatly but not totally relieved by an artificial eye. The latter is a cosmetic substitute which gives greater or less, never entire, satisfaction. Some people cannot wear an artificial eye at all,—it causes too much pain and secretion; in many others it fits and moves badly on account of cicatricial bands which form immediately after the operation if the eye was diseased, or later by ulceration, thickening, and contraction of the conjunctiva from carelessness of the patient, or chronic conjunctivitis, dacryocystitis, etc., so that the artificial eye becomes more and more troublesome and has to be left off altogether. The operations to model a stump when the conjunctiva is shrunken are mostly unsatisfactory. I should not omit to mention, however, that in the *majority of cases* an artificial eye is a tolerably good cosmetic substitute, and can, with proper care and cleanliness, be worn without notable discomfort for many years, even for a lifetime.

The *mortality from enucleation is very low*, certainly not more than one in five hundred or one in a thousand. O. Becker[1] has compiled one thousand cases without a death, and H. D. Noyes reports[2] eleven hundred and sixty-four cases from his own practice without a death, of which fourteen per cent. were of panophthalmitis. I have made more than one thousand enucleations; none has terminated fatally.

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[1] O. Becker, Die Universitätsaugenklinik zu Heidelberg, 1888.

[2] H. D. Noyes, Enucleation during Panophthalmitis Suppurativa, Transactions of the American Ophthalmological Society, 1889, p. 314.

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ENUCLEATION IS INDICATED:

(1) In *intolerable pain with incurable blindness.* Once a patient on leaving the hospital said to me, "Now I go home a happy man," after I had removed his *second* eye. "I knew I had to remain blind," he added. "I am happy to be no longer on the rack." I consented reluctantly to do the operation, but his satisfaction after it showed me that human happiness, after all, is only relative.

(2) In pain, inflammation, hemorrhage, photopsia, and other irritative symptoms, in chronic *irido-choroiditis, glaucoma, phthisis bulbi,* etc., in one eye which is blind or surely will be blind, irrespectively of the other.

(3) In eyes so *extensively injured* that a recovery is not to be thought of.

(4) In *intra-ocular malignant tumors,* gliomas and sarcomas, except when they are small and situated in the iris, so that they can be radically removed.

(5) In *epi-ocular malignant tumors,* if they cannot be removed without destroying the eye.

(6) In *orbital tumors* that threaten life, if it is impossible to extirpate them without sacrificing the eye.

(7) In *staphyloma* and *macrophthalmus,* if the eye is blind and becomes unsightly and troublesome.

(8) In *panophthalmitis,* conditionally. Compare page 887, etc.

(9) In the presence of foreign bodies in the eye that cannot be removed and cause trouble, inflammation, and blindness.

(10) In traumatic irido-cyclitis to prevent or cure *sympathetic ophthalmia.*

Sympathetic ophthalmia is an irido-cyclitis plastica or serosa, transmitted in an unknown manner from the fellow-eye in which an injury of the iris and ciliary body previously had caused the same disease. The sympathetic inflammation rarely shows itself in the first month after the injury, less rarely in the second and third, most frequently in the fourth and fifth, and then more and more rarely in the following months or years. Children are more apt to be affected with it than adults; the winter is more favorable for its development than the summer; exposure, carelessness, and taxing the eyes during the course of the inflammation in the injured eye are known to have preceded the development of sympathetic ophthalmia. For years I have watched with particular attention the penetrating wounds of the cornea and ciliary region, and have never seen that they were followed by sympathetic ophthalmia unless the iris or the ciliary body were cut or lacerated; the mere prolapses of these parts, if they were left alone and protected from injury, did not produce sympathetic ophthalmia. It is well known that sympathetic ophthalmia occurs more frequently after the combined than after the simple extraction of cataract. Arlt says,[1] "Cases of sympathetic ophthalmia are by far less frequent after flap extraction than after [the combined] peripheral linear extraction." This was written in 1874, when Graefe's extraction had almost universal sway. I remember several cases of sympathetic ophthalmia from combined extraction, none from simple extraction (in more than thirteen hundred cases), except two of complicated cataract with prolapse of the iris which I had cut several days after the operation.

[1] Arlt, Graefe-Saemisch, Handbuch der gesammten Augenheilkunde, Bd. iii. S. 424.

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Enucleation of the injured eye is the only remedy for sympathetic ophthalmia, and is reliable only as a prophylactic measure before the other eye is involved. The serous form of sympathetic ophthalmia affords a better prognosis, and may recover without or with removal of the exciting eye. The plastic irido-cyclitis begins with a faint red halo around the cornea, dulness of the iris, contracted, not fully dilatable pupil, and the appearance of exceedingly small, fine posterior synechiæ, which at the beginning are recognizable only with a magnifier. This condition may develop with or without any symptoms of irritation (photophobia and asthenopia). The latter symptoms—sympathetic irritation—are without any consequence if unaccompanied by the physical signs of iritis. The indication for enucleation, to my mind, is given much more by the condition of the injured eye than by any mere irritation of the other. It is exceptional to see sympathetic inflammation develop so long as the injured eye does not show a plain picture of irido-cyclitis. I watch both eyes carefully every day, treating the first as a grave inflammation in any case, but so long as its iris is bright, without circumcorneal injection and filiform adhesions around the pupil, and the other eye is free from the same changes, I do not think of an enucleation. If there is a distinct iritis, with still good sight, I do not at once propose an operation, for a traumatic iritis is usually recovered from, and rarely induces sympathetic ophthalmia. If, however, the iritis is progressive and assumes the picture which we are accustomed to see in sympathetic ophthalmia, I do not wait to advise enucleation until the fellow-eye is affected, but inform the patient or his relatives of the danger, telling them that sympathetic ophthalmia does not necessarily follow under these circumstances, but that if once begun, blindness of both eyes would be the almost certain issue. If they refuse to give their consent, I do not give the case up, but ask them to consult another oculist, and treat the patient to the best of my ability. The symptoms of fully developed irido-cyclitis which signalize danger from sympathy are: pain with nightly exacerbations, diminution of sight, circumcorneal injection, dull, drab-colored, uneven, slightly nodular, swollen, and irregularly bulging iris, posterior synechiæ increasing steadily to form a pupillary membrane, punctate deposits on the walls of the anterior chamber, vitreous hazy, fundus veiled, its details unrecognizable in the advanced stage, field of vision entire or defective, the latter especially if the injury was done by a foreign body still in the eye, ciliary region, particularly its upper part, tender on pressure, in the first stage transient increase of eyeball tension, or alternation of + and — T, later on permanent diminution, ultimately blindness, which, however, may require years to be complete. As soon as this picture is declared—long before it has run its course, and before any abnormity in the other eye is discoverable—I urge enucleation, and, if refused, throw the whole responsibility on the patient, not afraid of my reputation in case the eye recovers without an operation, for there is imminent danger of incurable blindness in both eyes, and we as physicians are

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not allowed to let the patient incur such a risk without informing him of it. If the injured eye is blind, or going steadily from bad to worse, there is no doubt what should be done; but another question arises which is more difficult to answer.

What shall we do in case the injured eye has still useful sight when the disease appears in the other? In this case I would no longer advise an operation, for the latter is almost always without any influence on the ultimate fate of the sympathetically affected eye (a temporary improvement immediately after the operation usually disappears in a week), and not a few cases are on record—I have seen some in my own practice—where the injured eye recovered sight, whereas its fellow became blind.

In *making the diagnosis of sympathetic ophthalmia* we should distinguish between an ordinary, not sympathetic iritis and the peculiar irido-cyclitis described above. This is of importance when iritis affects a previously good eye whose fellow is degenerated and sightless. In such cases, where others wanted to enucleate the blind eye, I had the satisfaction of seeing the eye recover from its iritis without my interfering with the other. I do not think it of any use to remove such an eye in the expectation of making an operation, say for cataract on the other eye, safer, provided the sightless eye is and has been for a longer time free from irritation.

If the injured eye is blind and not yet free from irritation when sympathetic ophthalmia affects the other, we should enucleate the former, for it is conceivable that the injured eye, as it did at first, may continue to induce a morbid process in the other. Experience, unfortunately, shows that the removal of the offending eye, even if done at the outbreak of the sympathetic ophthalmia in the other, has very little, if any, influence on the final result of the disease in the latter.

Yet it should be borne in mind that *not all eyes affected with sympathetic inflammation become blind;* there are milder forms, the more serous inflammations, in which optic neuritis sometimes is a prominent symptom, which give, as I have stated above, a better prognosis.

II. EVISCERATION OR EXENTERATION OF THE EYEBALL.

Careful cleansing (asepsis) always presupposed, the lids being separated with a speculum, the operator steadies the eyeball with fixing forceps implanted near the lateral corneal limbus: an assistant may do the same five millimetres backward. The operator incises the sclerotic between the two pairs of forceps, near the one implanted in the sclero-corneal border, with a scalpel, cautiously, by successive strokes until the pigmented ciliary body is laid bare. One branch of strong scissors with slender and blunt-pointed tips (Stevens's) is introduced through the opening and slowly pushed through the suprachoroidal space, the point sliding against the sclerotic, not against the ciliary body, for three or four millimetres parallel with the corneal margin, when the sclerotic comprehended between the branches of the scissors is cut. The lips of the end of the wound are now separated by the two

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pairs of forceps as before, the inner branch of the scissors advanced farther, and the sclerotic cut. This manœuvre is repeated until one-half of the sclerotic is incised. The other half is divided in the same way, beginning at the temporal side, and going around until the two incisions meet. The wound is kept open with two pairs of forceps at the upper edge about ten millimetres apart, and a flat, sharp, broad spoon (the curved hand-chisel (Fig. 21) for the removal of cystic tumors will answer) is pushed between choroid and sclerotic, always resting on the latter, carried sideways and deeper to detach the whole contents of the eye from the sclerotic, if possible unbroken. The vorticose veins and the optic nerve offer the greatest resistance, and must be severed with particular care. The inner side of the sclerotic is inspected, and if any particles of tissue are left they must be cleanly removed with a sharp spoon or a curved chisel, and in some cases, moreover, with sterilized gauze. The cavity is then irrigated with a mild antiseptic and allowed to fill with blood, if there is a sufficient flow. The wound is closed with four or five sutures passing through the conjunctiva and the edges of the sclerotic. The circular form of the wound becomes linear, with projecting corners, unless we remove small triangular pieces at the ends, as in the amputation of corneal staphyloma (see above), which is advisable.

If it is not possible to evacuate the contents of the globe as a whole, they must be removed piecemeal by cutting, scraping, and wiping until the inner surface of the sclerotic is perfectly clean. Excessive intra-ocular hemorrhage may require packing the wound-cavity with gauze and a firm pressure-bandage upon the lids and orbit.

The recovery is commonly less smooth than that from enucleation. There are some pain, secretion, and swelling of lids and conjunctiva. The eyeball fills with blood, hardens, and protrudes in consequence of œdema of the orbital tissue caused by thrombosis of the vorticose and orbital veins.[1] There may be mental apathy, even drowsiness, for some days. Commonly all these symptoms disappear in two or three weeks. In many cases there is no noteworthy reaction. The sutures are removed in from five to eight days. An artificial eye should not be worn before a month.

The operation may be *varied* as that of staphyloma; which compare. For the delicate circumcision in the ciliary region the simpler abscission of the cornea at the limbus may be substituted, which has the advantage of preserving the corneo-scleral border and leaving a larger stump. This modification may replace the above-described method in cases where a satisfactory examination does not require the integrity of the specimen, for instance, in panophthalmitis, etc.

Prince (1888) paints the inner surface of the sclerotic with a ninety-five per cent. solution of carbolic acid, which, he alleges, diminishes the reaction.

[1] See H. Knapp, Case of Evisceration of the Eye, followed by (Non-Purulent) Orbital Cellulitis (Thrombosis); Recovery. Remarks. Archives of Ophthalmology, vol. xiv., 1885, p. 309, and Archiv für Augenheilkunde, Bd. xvi., 1886, S. 55.

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He packs the wound-cavity with iodoform gauze, which he leaves for weeks until the granulations cast it out.

INDICATIONS AND RESULTS.

Evisceration, according to its ardent advocates, should be substituted for enucleation in all cases except in intra-ocular tumors and foreign bodies. Other authors, the great majority, except also the cases of sympathetic ophthalmia, because the parts from which the transmission of the disease may take place are more thoroughly removed by enucleation than by evisceration; besides, there are cases on record in which sympathetic ophthalmia occurred after evisceration.[1]

In the case of Cross sympathetic ophthalmia set in four months after the injury, and three weeks after the evisceration; in the case of Hotz the injury was several years, the evisceration seventeen days previously. Czermak[2] says, "These cases are not conclusive, for the transmission may have been present before the operation." This is true; but similar examples after enucleation are on record. The greatest objections to evisceration are, first, that the stump for prothesis is soon not much larger than that after enucleation; and, secondly, that enucleation is easier to perform and more readily recovered from than evisceration. Yet the stump after evisceration *is* larger, for there is more substance, the whole sclerotic, in it, and the recovery, even if more protracted, is not very long, and seems to be about as safe.

The present writer has not been partial to evisceration, but feels inclined to substitute it for enucleation in a greater percentage of cases than he cared to do formerly. Dr. Bunge writes him that Alfred Graefe and himself have had no case of death after their about five hundred eviscerations. He continues to perform enucleation as he described it in his monograph.

III. EVISCERATION WITH INSERTION OF AN ARTIFICIAL VITREOUS.

Mules's Operation.[3]—Immediately after the stoppage of the hemorrhage and cleansing of the intra-scleral cavity a bead of glass (Mules), celluloid (Lang), silver (Kuhnt), or some other substance is inserted to fill the cavity so that it may be closed by sclero-conjunctival sutures without stretching.

To facilitate the insertion of the glass bead Mules had an *instrument* (see Fig. 41) made, consisting of elastic narrow metal bands projecting from the edge of a tube and so bent as to form a sphere, a neck, and a cup. The ball is put into the cup, pressed through the neck, and received at the bottom of the sphere by a concave disk fastened upon a rod that runs through the cylinder and projects beyond it from one and one-half to two

[1] Cross, Ophthalmic Review, 1887, p. 693; Journal of the American Medical Association, 1893, October 31.

[2] Czermak, Die augenärztlichen Operationen, S. 443.

[3] Mules, Evisceration of the Eye and its Relation to the Bacterial Theory of the Origin of Sympathetic Disease, British Medical Journal, 1886, vol. i. p. 246.

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centimetres. When the glass bead is in the sphere, the tips of the elastic bands are introduced into the scleral wound, and the bead is pushed into the scleral cavity by pressing upon the knob of the rod.

Fig. 41.

Inserter of glass bead.

I have made a few of these operations. The bead healed in nicely and without reaction, and the artificial eyes fitted and moved perfectly and caused no discomfort, but I cannot tell how long this most satisfactory condition lasted, for I lost sight of the patients.

Upon inquiry during my travels in Europe as to the tolerance of the eye for the large foreign body, I heard more unfavorable than favorable reports. In a small percentage of the cases the bead was not tolerated at all; in many it had been borne with comfort for a number of years, but then was discarded. Czermak[1] says, "Mules's operation has proved a failure. Such foreign bodies are all cast off sooner or later." Yet the operation has continued in favor. Its most ardent advocates at present are Mr. J. H. Bickerton, of Liverpool, and L. Webster Fox, of Philadelphia.

ARTIFICIAL EYES (PROTHESIS).

Artificial eyes are now made in all civilized countries. Their value is more than cosmetic. Absence of the eyeball, or a very small stump, causes sinking and inversion of the lids, especially the upper, lacrymation, and muco-purulent discharge, also excoriation of the lids. All these symptoms disappear after proper prothesis.

The insertion of an artificial eye requires an adequate free space behind the lids. To widen an insufficient conjunctival sac (see above, operations on the conjunctiva) is a most difficult and usually a thankless task.

To insert and remove an artificial eye is easy, and soon learned by the patient when he sees it a few times done by the manufacturer or the oculist. The cleansing of the artificial eye is important. It should not be inserted too soon after the operation, and should always be left off when there is conjunctival inflammation. It should be taken out before the patient goes to bed, put into water, and washed with soap; from time to time also put in alcohol. As soon as it becomes corroded, rough, it should be replaced by a new one, for rough eyes irritate and inflame the stump, which causes secretion, hyperplastic inflammation, and subsequent shrinkage of the conjunctiva, so that an artificial eye can no longer be worn with comfort, and

[1] Czermak, Die augenärztlichen Operationen, S. 447.

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ultimately not at all. As soon as conjunctival inflammation sets in it should be carefully treated with antiseptics and astringents.

Exenteration of the orbit will be described later.

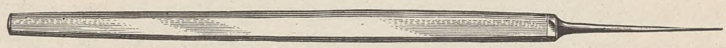
§ IX. OPERATIONS ON THE TEAR-PASSAGES.

(*A*) OPERATIONS ON THE PUNCTA AND CANALICULI.

I. DILATATION OF THE LACRYMAL PUNCTA.

This little operation is done with a *conical probe.* (Fig. 42.) The lid is turned outward and steadied by drawing it towards the lateral orbital

FIG. 42.



Conical probe to dilate lacrymal punctum.

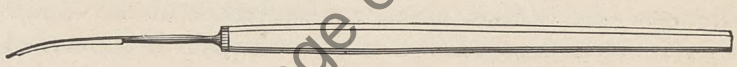
wall. The point of the probe is first directed perpendicularly to the edge of the lid, but as soon as it has entered the canaliculus it is directed towards the inner canthus and advanced in the canaliculus as far as the intended dilatation requires. In one or several minutes it may be withdrawn.

Indications.—This little procedure serves chiefly as a preparation for rinsing the lacrymal passages with a narrow-nozzled syringe (Ed. Meyer's, see Fig. 44, p. 896) or for slitting the canaliculi.

II. SLITTING THE CANALICULI.

Technic.—The operator may stand behind the patient, holding the latter's head against his breast, or he may stand or sit before the patient, whose head is steadied by an assistant. If the lacrymal point is narrow, it should be dilated as described above; if of sufficient size, the operator introduces the blunt-pointed tip of a Weber canaliculus knife (Fig. 43)

FIG. 43.



Weber's canaliculus knife.

into the canaliculus of the stretched lid, advances it as far as the caruncle, and turns it on its tip so as to split the inner-upper wall of the lower, or the inner-lower wall of the upper, canaliculus. There is very little bleeding, and the patient may immediately go home or to his work. The wound will unite at once, but has to be opened with a probe daily for several days, until each of the two lips is covered with epithelium and a permanent slit in the canaliculus secured.

Indications and Results.—Slitting of the canaliculi is *indicated:*

(1) In eversion of the puncta, especially the lower, for epiphora and its

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consequences, excoriation of the lid, etc. This unpleasant condition is mostly relieved by the little operation. Arlt and other authors affirm that splitting of the canaliculi in their external (temporal) part does not diminish the absorption of the tears, which I doubt: it may not make it impossible, and may favor it in cases of eversion, but I should prefer to preserve my natural puncta and canaliculi.

(2) As a preparatory operation for syringing, probing, and scraping the tear-passages.

(3) To remove polypoid growths or fungous and calcareous collections in the canaliculi. The calcareous masses (lacrymal calculi) are calcified fungous growths (actinomyces, formerly considered to be leptothrix), and show as round or oval swellings in the canaliculus. They are partially evacuated through the tear-points on being compressed. After slitting the canaliculi they can be easily and thoroughly evacuated, but, as I have convinced myself in a recent case, they can also be radically removed without splitting the canaliculus. It suffices to dilate the punctum, press the mass out as much as possible, then throw into the canaliculus with a fine syringe (Ed. Meyer's) an antiseptic solution (a four per cent. alcoholic

FIG. 44.

Ed. Meyer's lacrymal syringe.

solution of salicylic acid) daily until all secretion, swelling, and irritation have disappeared, which may take a week or two.

III. OBLITERATION OF THE LACRYMAL POINTS AND CANALICULI.

Technic.—The puncta and canaliculi are closed with the galvano-cautery or by a ligature which constricts the canaliculus near the punctum.

Indications.—In injuries, ulcers, and operations of the cornea when dacryocystitic secretion prejudices a smooth recovery. If the corneal affection is transient and not very severe, temporary obliteration may suffice. This is effected by introducing the electrode of the cautery only a few millimetres, or by tying the ligature so moderately as not to cut through, for instance, in cases of cataract extraction.

When the wound is firmly and permanently closed, say in from five to seven days, the ligature can be removed, and about a week later the canaliculus can be probed if it does not spontaneously become pervious again. This holds for the obliteration with either the ligature or the cautery.

If we want to produce a permanent obliteration, the electrode should be introduced about five millimetres and the ligature tightened so as to destroy the tissue at the place of the ligature.

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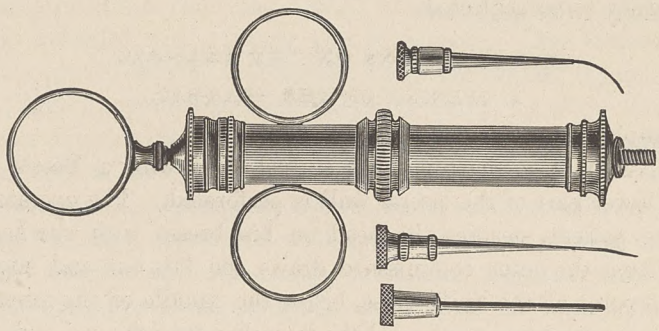
IV. SYRINGING OF THE CANALICULI AND THE OTHER PARTS OF THE TEAR-PASSAGES.

Besides a delicate syringe like E. Meyer's, the well-known larger one of Anel (Fig. 45) is used for syringing the tear-passages. It has been in general use since 1716, and is at present as popular as it ever was. Its

FIG. 45.



Anel's lacrymal syringe.

cylinder is of glass. It has several nozzles, straight and curved, larger and smaller ones, which can be selected according to the case. They require a slit-up canaliculus, whereas Meyer's can be introduced through the natural or dilated punctum. A nozzle small enough for that purpose can, of course, be added to the Anel syringe.

Technic.—The canaliculus is stretched as in the operation of splitting. In most cases the nozzle of the syringe need be introduced into the canaliculus only,—*i.e.*, no farther than about the caruncle. The pressure in syringing should under all circumstances be light or moderate. The water may, 1, regurgitate through the same canaliculus, owing to a fold in the canaliculus, which can be smoothed out by better straightening the canaliculus, or by permanent occlusion of the canaliculus; 2, it may escape through the punctum of the other canaliculus,—closure of the common entrance of the canaliculi into the sac; 3, it may distend the sac,—occlusion of the naso-lacrymal canal; 4, it may pass into the nose,—perviousness of the whole passage.

In some cases the nozzles, both large and small ones, may be introduced into the tear-sac, and even through it into the nasal duct. Besides this, syringing may be done through a natural fistula or an artificial opening of the tear-sac through the skin or the conjunctiva.

Indications and Results.—Syringing is used:

(1) For *diagnostic purposes* in *obstructions*, as mentioned above, and in *inflammations:* the purity of the escaping water or its admixture with other substances will aid in determining the nature of the inflammation, whether catarrhal, purulent, or septic.

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(2) For *therapeutic purposes:* injection of sterilized water for cleansing, of antiseptics and astringents for inflammations.

It is prudent not to use the syringe in acute suppurations, nor in fresh wounds or after forcible probing, lest a severe purulent inflammation ensue, which may lead to abscess in the orbit and its consequences.

The beneficial action of syringing in relieving or curing inflammations of the tear-passages and their consequences is marked in many cases, and often superior to that derived from the customary probing. Syringing seems unjustly to be neglected.

(*B*) OPERATIONS ON THE TEAR-SAC.

I. OPENING OF THE TEAR-SAC.

(*a*) **Simple puncture, or paracentesis.**

Technic.—With a sharp-pointed scalpel, best with a Beer's cataract-knife, the lower part of the lateral wall is perforated. The operator stands behind the patient, steadies the head on his breast with one hand, with which he from the outer commissure draws the lids out and slightly up, thrusts the point of the knife close below the middle of the inner canthal ligament through skin, muscle, and the lateral wall of the sac, and pushes it on obliquely inward, backward, and somewhat upward until, by the escape of the contents of the sac or the resistance of the medial wall, he knows that the knife has entered the sac. On withdrawing the knife he enlarges the opening down and slightly outward as far as the orbital border. The puncture can be done as well from below upward.

Indications and Results.—The simple puncture is indicated:

(1) In *phlegmonous inflammation.* In the beginning, when the hardness, swelling, and redness are uniform, poulticing and compressive bandages may be beneficial, and in exceptional cases abort the inflammation, but as soon as there is the least fluctuation, softening, and pallor in some place, the phlegmon should be lanced. Even before these symptoms have appeared, when there is great pain and the above treatment does not prevent the increase of the swelling, the paracentesis of the sac should not be postponed, for it at once relieves the pain, the inflammation subsides, and recovery takes place more rapidly and with less disfigurement (mostly without any) than if the disease is left to run its natural course. In the latter case the destruction of the mucous membrane, the sac, and the skin is more extensive, and proportionately also the scar in the mucous membrane and the skin. If the phlegmon is primary, not grafted on old lacrymal disease, the recovery is commonly perfect,—*i.e.*, without a mark in the skin and without lacrymation.

In the after-treatment poultices and compressive bandages are of use, due consideration of the cause and complication of the affection being presupposed.

(2) In *mucocele, i.e.*, the distention of the lacrymal sac by a collection of sero-mucous fluid, for diagnostic purposes or for the application of local

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medicinal or surgical treatment, even for the mere relief of the patient, if the tumor be large, preparatory to more thorough measures, such as partial or total removal of the sac.

(3) As a *prophylactic* in ulcers, injuries, or operations on the cornea in chronic dacryocystitis with infective secretion. In this case the interior of the sac is sterilized, cauterized, and plugged, until the secretion has ceased.

(*b*) **Extensive opening of the sac.**

The *technic* is the same as in paracentesis, but the incision is larger. When the puncture is made and carried down to the lower orbital margin, it can be extended with a strong pair of scissors. One branch is introduced through the opening and pushed under the canthal ligament upward, curving slightly outward, as far as the top of the cavity of the sac. Closing the scissors divides the skin, the canthal ligament, and the upper part of the lateral wall of the sac.

The extension, however, can also be made with the knife, which, when it has touched the lower orbital wall, is turned so as to divide the ligamentum canthi and the wall of the sac in an upward, slightly curved direction. The same extensive incision can be done with one stroke of the knife from above down, with a slight concavity towards the cornea, following the course of the fibres of the orbicular muscle.

Indications.—This operation is done (*a*) for *removing foreign bodies* from the sac and the nasal canal, and (*b*) *preparatory to treating or obliterating the lacrymal sac.*

To treat the interior of the lacrymal sac in order to *restore* as much as possible its *normal anatomical condition and function,*

(1) *Caustics of different kinds and strength are applied.* The best is nitrate of silver, one per cent. or stronger. This is brushed over the whole extent of the mucous membrane and then washed off as in purulent ophthalmia. It acts also on the mucous membrane of the nasal duct, and can be repeated daily or every few days, and the wound kept open by packing it with sterilized gauze until the secretion has stopped and the mucous membrane is smooth.

(2) The mucous membrane, if it is granulating and has polypoid excrescences, is *curetted in all its diseased parts with a sharp spoon,*[1] and afterwards treated with *antiseptics and caustics.*

(3) The *cautery* (actual, thermic, or galvanic) is so highly praised by Panas that I shall translate what he says of its application and effect:[2] "In deep havoc of the sac, with or without fistula and bareness of the bone, the opening of the whole field, the division of the orbicularis tendon included, and the application of the thermo-cautery constitute the sheet-anchor of the treatment (*la pratique par excellence*). For twenty years that

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[1] Mandelstamm, Centralblatt für praktische Augenheilkunde, 1879, S. 179; de Wecker, Le raclage du sac lacrymal, Archives d'Ophtalmologie, t. xi. p. 494, and others.

[2] Panas, Traité d'Ophtalmologie, p. 350, last line, *et seq.*

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I have adopted it it has rendered me the greatest service. But the little cautery, already known to Paulus of Ægina, must have an olive-shaped tip about six by four millimetres in size. Heated to dark red, it should be passed over all points of the sac, on the side of the orifice of the canaliculi as well as on the nasal canal. Contrary to expectation, this treatment is purely cauterizing, and not at all destructive. After the cauterization the sac is plugged with iodoform gauze, which is left in two or three days, after which the cavity is irrigated antiseptically and filled each day with a pledget of gauze covered with a salve of binoxide of mercury. The suppuration soon stops, the sac closes in two or three weeks, there is no more discharge, and the lacrymation disappears or is considerably reduced. To dry it up completely, the lower canaliculus, the perviousness of which is of great importance, is slit open, and a Bowman probe, No. 2 or 3, passed through it a certain number of times. If there is a fistula, it obliterates; and as to the reproach of leaving a conspicuous cicatrix, it is entirely unfounded. Concerning the results I can affirm that they are excellent."

Variation of the Mode of Opening the Lacrymal Sac.—Instead of opening the sac through the skin, many operators have done it through the conjunctiva, by splitting the canaliculi in their whole length and extending the incision up or down to make it sufficiently large not only for evacuation of pus and mucus, but also for chemical and surgical applications. I do not think that the opening through the conjunctiva has any advantage over the opening through the skin. On the contrary, it endangers the eye by pouring the often infective contents of the sac directly over the cornea. The opening through the skin in itself leaves no notable scar, whereas the splitting of the inner wall of the sac through the canaliculi leaves a gap which is not only visible but attracts attention by being filled with tears. Cases of that kind which I have seen did not encourage me to make attempts in this direction.

II. PARTIAL EXCISION OF THE LACRYMAL SAC.

This operation has been recommended by Bowman, Despagnet,[1] and others in cases of voluminous mucocele. Bowman makes a large incision in the anterior wall and removes a portion of it. Despagnet opens the sac by a large external opening and removes a semilunar portion. He cuts off with scissors prominent vegetations, scrapes the walls with a half-sharp spoon, injects bichloride of mercury ($\frac{1}{1000}$), and touches the mucous membrane with a cotton pellet dipped in a mixture of glycerin and bichloride of mercury ($\frac{1}{200}$). This treatment is continued daily until the wound is closed (in eight or ten days). After this the sac is syringed out through the puncta. Despagnet reports radical cure in three chronic cases where probes and injection had been of no use. This operation seems beneficial in a goodly number of cases, but has never found sufficient endorsement by the profession.

[1] Despagnet, Bulletins et Mémoires de la Société française d'Ophtalmologie, 1891, p. 39.

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III. EXTIRPATION OF THE SAC.

This operation was first done by Platner in 1724. It has at times been very popular, at others severely criticised. It is done in two ways,—viz., with and without opening the sac.

(*a*) With *opening the sac*, apparently the more common method. The patient is narcotized. The sac is opened in the manner described above, emptied, and wiped out. Profuse hemorrhage is usual in this operation, but can be avoided or restricted by sparing the angular artery and vein, pressing the skin against the bone of the nose, and careful wiping. It is prudent before beginning the separation of the wall of the sac to ascertain the position of the orifices of the canaliculi, and especially the beginning of the nasal canal, by probing. The wall of the sac has to be dissected from the skin and then from the bone by fine scalpels, scissors, and slightly curved sharp or semi-sharp hand-chisels. The skin is drawn to the side by clawed retractors or specula. It is best to begin with the medial wall, where the sac is intimately connected with the periosteum, then advance towards the cupola and the lateral wall, then separate the sac from its posterior support, and so down to its entrance into the nasal duct, where it should be carefully isolated and cut close to the bone. As soon as the bleeding has sufficiently subsided, an oblong sharp curette is used to scrape the nasal duct down to its orifice in the lower meatus.

To do this operation cleanly is tedious and difficult. Hastening and not minding the bleeding lead to a matting together of the parts, cutting away the surroundings of the sac on one side and leaving portions of it behind on another. The frequency of the latter condition has led to

(*b*) *The method of extirpating the sac without opening it*, which, if it succeeds, is decidedly to be preferred. It requires perhaps a little more patience and delicacy than the other method. The integument is divided layer by layer, in the same curvilinear direction as in the other method, until the anterior wall of the sac is exposed. The walls of the sac are separated from their surroundings as above described. If the sac is not perforated accidentally, its contents may escape through the division of the canaliculi, and they surely will do so when the union of the sac with the periosteum at its entrance into the nasal duct is divided.

As *accidents during the operation* the following may be specially mentioned.

(1) *Inundation of the eyeball and the wound with the contents of the sac.* As these are frequently infective, the operation should not be undertaken when injuries or ulcers of the cornea are still present, unless they are infected themselves and their recovery is prevented by the discharge from the lacrymal sac. Accidental scratching or other injury of the cornea during the operation must be carefully avoided. Thorough though gentle sterilization of the wound and the conjunctival sac should be attended to before the dressing.

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(2) The *bleeding* during the operation is sometimes so troublesome that patience and wiping should be supplemented by instrumental compression and ligation, which mostly need be only temporary. The bleeding has led to the *modification of performing the operation in two sittings*, in the first to split the sac, scrape away its contents and any depositions and granulations on the wall with a sharp spoon, and pack the cavity with antiseptic gauze for a day or two, then remove the packing and complete the operation as above described.

(3) *Leaving parts of the sac behind*, the most serious mistake, would baffle the object of the operation by partial restoration of the sac and return of the suppuration with its consequences. Such portions have all to be removed with scissors or a sharp spoon before the wound is dressed.

After-Treatment and Course of Healing.—The wound is united by deep sutures, covered with sterilized gauze and cotton held by a compressive bandage. In from four to six days the sutures are taken off. Healing by first intention, terminating in recovery in from ten to fourteen days, is the rule. No scar conspicuous in any way is left, unless complications of fistulous passages with either indrawn cicatrices or hyperplastic granulation-tissue had been present before. All these complications should, of course, be removed before or during the operation. In dyscrasic subjects, especially when complications, above all caries, are present, suppuration may follow the extirpation of the sac, with erysipelatoid swellings of the lids and with orbital cellulitis, which in exceptional cases may be followed by optic nerve atrophy; even death from thrombo-phlebitis and meningitis cannot absolutely be excluded. Sloughing of the cornea and panophthalmitis are also mentioned as possible consequences of the operation, but they are very rare.

Indications.—*Extirpation* of the tear-sac is *indicated when an important lacrymal disease can otherwise not so well or not at all be cured.* This is the case with the following conditions:

(1) *Large mucocele*, chronic distention of the sac by sero-mucous fluid.

(2) Chronic *lacrymal fistula*, excepting the capillary fistula.

(3) Fibrous or osseous *occlusion of the nasal canal* with troublesome lacrymation.

(4) Chronic *stricture of the nasal duct* with lacrymation and discharge.

(5) Chronic *dacryocystoblennorrhœa* with degeneration of the walls of the sac.

(6) Chronic *catarrhal dacryocystitis* with repeated attacks of *acute phlegmonous inflammation.*

Extirpation of the sac is as radical an operation as an amputation, which, I repeat, should not be resorted to unless other modes of treatment have failed. A part of those modes of treatment have been described in the previous pages (see the indications for opening the lacrymal sac, etc.); the other part will be described later (see operations on the nasal duct).

The same object, to cure a troublesome and dangerous lacrymal disease, may be attained not only by extirpation, but also by

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IV. OBLITERATION OF THE TEAR-SAC BY CHEMICAL OR PHYSICAL MEANS.

Injection of tincture of iodine, which formerly was recommended, acts beneficially on the diseased mucous membrane of the sac, but will in itself not obliterate the sac. *Caustic alkalies* and *strong acids* will destroy the walls of the sac and obliterate the latter, but their action is too violent and often destroys more than is intended. *"The best remedy is nitrate of silver"* in substance, says Arlt,[1] and that it acts well I can confirm from personal experience, "but," adds Arlt, "a single application will not do," which I can also confirm.

Technic.—The sac is opened by a large incision. After stoppage of the hemorrhage the wound is held open by retractors, wiped dry, and cauterized freely with the nitrate of silver crayon from the cupola down to the beginning of the nasal duct, so that no point of the sac-wall remains untouched.

Arlt recommends to do the operation in two sittings, of which I heartily approve, though I have not tried it. The bleeding and moisture before, during, and after the cauterization are troublesome. Arlt opens the sac largely, stills the bleeding, and packs the sac with lint or a pressure sponge. When he removes the packing on the second or third day, he has a large, dry cavity before him, an ample compensation for the delay. He applies the nitrate of silver as above described and bandages the eye. As soon as the eschar loosens, it is removed, the sac cauterized again, and a new plug put in. This is repeated as long as there are places of mucous membrane still covered with epithelium. To obliterate the sac completely may take several weeks, even a month or two. The treatment is tedious, but not particularly unpleasant. The bandage may, after the second cauterization, be replaced by a single layer of isinglass plaster. No conspicuous scar is left.

The sac may be obliterated with an olive-shaped tip of the *thermo-* or *galvano-cautery,* applied in the same way as the nitrate of silver stick, mostly also requiring more than one sitting. To dilate the wound by packing it for a day or two will here also be of advantage.

Results.—These operations are more tedious than the extirpation, but appear less terrible to the patient, and, so far as my experience goes, they are not so dangerous, suppuration and orbital cellulitis being less frequent.

Their *value* is very great. A disease (for instance, dacryocystoblennorrhœa with fistula, which gives the strongest and most frequent indication) dangerous to the vision and, though rarely, to life, but irksome under all circumstances, is eradicated, and leaves nothing but slight lacrymation in cold and windy weather, and even that, if troublesome enough, may be cured by removal of the lacrymal gland.

Now and then attempts to cure epiphora and discharge by obliteration

[1] Arlt, Graefe-Saemisch, Handbuch der gesammten Augenheilkunde, Bd. iii. S. 497.

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of the sac have failed to obliterate the sac, but led to a much more satisfactory condition, the restoration of the structure and function of the tear-passages. Desmarres, who was the greatest advocate of obliteration, says that after complete obliteration no epiphora is left. Arlt says that in cases where obliteration was tried and no lacrymation was left, the exact physical and functional examination had demonstrated the restoration of normal perviousness and function of the organs. This is especially noticed if a degenerated (swollen and granulating) mucous membrane after the opening of the sac was cauterized with the nitrate of silver stick, just as it explains the remarkable and very suggestive statement of Panas, of which I have given a full translation above (p. 899). In obstinate, not to say desperate, cases of dacryocystitis, we may open the sac and, according to the condition of the mucous membrane, use the chemical or the thermic cautery with a view to cure the disease, or, in case of failure, obliterate the sac. The facts above described warrant an exploratory opening of the sac, cauterization of the mucous membrane without destroying it, and watching the effect. If the improvement after the first cauterization gives us a reasonable hope of restoring the function of the organ, the patient may in case of success thank us for the time and trouble the treatment has required, whereas in the other case we may let the second and third cauterizations follow the first at short intervals until the obliteration is complete.

(*C*) OPERATIONS ON THE NASO-LACRYMAL CANAL.

I. SYRINGING.

See above, under "syringing of the canaliculi and the other parts of the tear-passages" (p. 897).

The chief operation is

II. PROBING OR CATHETERISM OF THE LACRYMAL PASSAGES.

The calibre of the probes has to be suited to the *dimensions of the different parts of the tear-passages.* These, according to Panas, are as follows:

	Millimetres.
Diameter of the upper punctum	0.25
Diameter of the lower punctum	0.33
Length of the canaliculi from the puncta to their common mouth into the sac	5–7
Diameter of the canaliculi	0.50
Vertical diameter of the tear-sac	12–15
Antero-posterior diameter of the tear-sac	5–6
Transverse diameter of the tear-sac	4–5
Diameter of the nasal canal	3

The canal varies according to the age and size of the individual. The direction of the nasal canal has a threefold curve, from above, in front, and in, to down, back, and out. The direction from in to outward—*i.e.*, away from the median plane—is the rule, but varies in different individuals,

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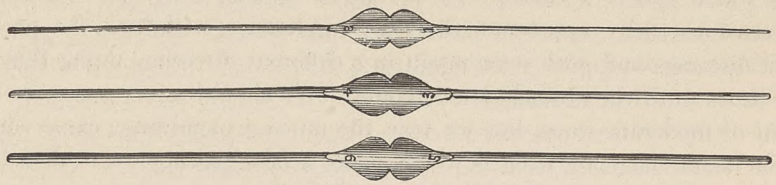
being sometimes parallel to the median plane, and sometimes even from above inward. This deviation is slight in any case.

The probes in common use are those of Bowman (Fig. 46). They are three double probes; each has a small plate in the centre, indicating the numbers, of which there are six, varying from one-half to one and a half millimetres in thickness, "reaching from a fine hair probe (No. 1)

FIG. 46.



Bowman's lacrymal probes.

to one of $\frac{1}{20}$ inch diameter (No. 6)."[1] The widths of those that are in common use are not exact, and vary greatly. To explore the canal, No. 3, equal to one millimetre, is the most useful; Nos. 1 and 2 are rather thin, and more liable to make a false passage than larger ones; Nos. 5 and 6 are so large that they do not readily pass through the passages at the first catheterization.

The probes are curved so that the concavity corresponds to the side of the little plate that bears the number.

Probing is difficult, and only No. 1 could be used if the punctum is not dilated. It may do for exploring the canaliculi, but not the nasal duct.

Technic.—The sac being emptied by pressure, the patient leans his head against the chest of the physician, who stands behind or sits before him, while an assistant steadies the head. He stretches the upper (or lower) lid with the index-finger of one hand towards the outer orbital margin, at the same time slightly everting the edge of the lid. Holding the probe between the thumb and the first two fingers of the other hand, he introduces the probe first one millimetre vertically to the edge of the lids, then medially through the whole length of the canaliculus into and through the sac until he feels the inner wall of the latter. Keeping the probe gently pressed against the nose, he raises it to a vertical direction and thrusts it downward, sliding over the medial wall of the sac until it slips into the nasal duct. He pushes the probe downward, never using much force, in a slightly outward direction, until he feels another bony resistance, the floor of the lower nasal meatus. The plate, in an adult, then is on the brow, and the upper half of the probe inclines a little towards the median plane of the body. In some cases it leans the other way, which may be correct, but is abnormal. The probe is slowly and gently withdrawn in from fifteen to sixty minutes.

[1] W. Bowman, On the Treatment of Lacrymal Obstructions, Royal London Ophthalmic Hospital Reports, London, 1857, p. 10.

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Difficulties, Accidents, and Mistakes in Probing.—There may be all kinds of obstacles in the way. The canaliculus may be strictured or occluded. It is not uncommon that folds of the mucous membrane, especially immediately before the common orifice, stop the advance of the probe, just as it does in the nasal portion of the passage, at the entrance of the sac into the canal, where the mucous membrane is so vascular as to resemble erectile tissue. The probe is readily engaged in the yielding structure, which then feels like a fold or a valve. To obviate or smooth away the impediment we must carefully appreciate the least hinderance, withdraw the probe a short distance, and push it on again in a different direction, doing this several times until the obstacle is overcome. We should never use more than slight or moderate force, lest we tear the mucous membrane, cause considerable hemorrhage, or, what is worse, make a false passage,—*i.e.*, detach the mucous membrane, which here is inseparably connected with the periosteum. Tearing of the mucous membrane and false passages readily cause inflammation of different degrees, acute hard swelling either disappearing by resolution or developing into an abscess which perforates the mucous membrane and empties into the conjunctival sac, or causes orbital cellulitis, which in rare cases is followed by optic neuritis and blindness, exceptionally by fatal meningitis.

In introducing the probe, we judge of its position by the movements of the skin at the inner commissure; if it moves with the probe, the latter is arrested before the common orifice of the canaliculi; to insure its passage we have to withdraw it a little way, stretch the canaliculus over it, and advance it again, also rotate it, until it reaches the nasal wall of the sac unobstructed. On moving the probe slightly back and forward, the skin no longer accompanies the movement.

The next and rather a frequent difficulty is the sliding of the probe over an elevation of the mucous membrane on the nasal wall of the sac, and falling on the opposite side, the lateral wall, close by the orifice of the nasal duct. Pushing the probe on just in this place is the most frequent cause of a false passage. If we feel the resistance we must not force the probe, but should draw it back to the nasal wall of the sac and push it down again, with rotatory and back or forward movements, but always gently sliding over the nasal wall of the sac. When we have made four or five unsuccessful attempts, we should draw the probe out, wait from fifteen to thirty minutes, and begin anew. Should this also be unsuccessful, we let the patient go home, to return in a few days. It is not rare that we try longer before we succeed. During the time we should endeavor to reduce the calibre of the mucous membrane by instillations of mild antiseptics into the conjunctival sac and washing out the lacrymal passages. In the repeated trials we may use fine and medium-sized probes, but never thicker ones than Bowman's No. 5, for fear of rupturing the united portion of the canaliculi, which may lead not only to stricture but to incurable closure of this narrow tube. If a thin probe passes, we introduce it daily

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for a few days, then every other day; then gradually we use thicker probes (but, in my opinion, which concurs with that of Arlt, Czermak, and many others, not beyond No. 5, or for large adults No. 6), which widen the canal sufficiently to restore and preserve the normal function of the tear-passages. When the canal is sufficiently wide, the probe may be passed once a month or at still longer intervals, so as to keep the passage sufficiently free until permanent recovery is secured.

In probing the lacrymal canal, as in catheterization of the urethra, nervous symptoms, such as fainting and epileptiform attacks, have been noticed. Rampoldi[1] reports ten cases in which probing caused mydriasis.

Indications and Results.—Probing is indicated in *strictures of the tear-passages during chronic and subacute inflammatory swelling of the mucous membrane.* It is a beneficial adjuvant in the treatment; no more. When in catarrhal dacryocystitis, or in the later stages of dacryocystoblennorrhœa, the secretion is pent up in the tear-sac, its removal by compression, incision, and syringing is of primary importance, for its presence will lead to prolongation and extension of the inflammation. Probing during the acute stage is dangerous, for the brittle or ulcerous mucous membrane, full of infective germs during the acute inflammation, is easily ruptured. Through the wound inflicted by probing the infective material is carried into the depth of the tissue by the natural currents of liquids, and still more by syringing and pressure of any kind. When, however, the acme in the inflammation is over, and the still engorged mucous membrane of the naso-lacrymal canal impedes the natural outlet of the contents of the tear-sac, judicious probing is very beneficial. It will be so in all stages of catarrhal dacryocystitis when from any cause the engorgement of the mucous membrane blocks or narrows the calibre of the canal. In chronic narrowing by cicatricial strictures, the conventional treatment by probing is a greater trouble to the patient than the evil. If it were conducive to a permanent recovery, the patient might well put up with the pain and annoyance it causes, but this treatment is at best only palliative.

CONCLUSION.

I advise patients with moderate epiphora to bear it without probing and attend to the conjunctiva and the mucous membrane of the nose. I treat acute diseases of the tear-passages antiphlogistically in the first stage and by cautious syringing and catheterization in the subsequent stages; leave incomplete chronic strictures alone; treat lacrymal fistulæ and chronic dacryocystoblennorrhœa by opening the tear-sac freely; pack it with sterilized gauze for a day or two, so as to make a thorough examination, and if I find the mucous membrane granulating, exuberant, I treat it with nitrate of silver in strong solutions or with the stick, or scrape and cauterize it, trying to restore its structure and function. Failing in this attempt, I obliterate or extirpate the sac.

[1] Rampoldi, Una nuova causa di midriasi, Annali di Ottalmologia, vol. xi., 1882, p. 513.

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There are a number of modes of treatment recommended for stricture of the nasal canal,—cutting, forcible dilatation with large probes, scarification, electrolysis, etc., of which some are still on trial; the others have not survived the trial.

§ X. OPERATIONS ON THE CONTENTS, WALLS, AND ACCESSORY SINUSES OF THE ORBIT.

I. EXPLORATORY OPERATIONS.

In the orbit, as in almost every other field of surgery, exploratory operations are of great value. Whether a certain intumescence contains blood, serum, mucus, pus, or soft, and even hard, tissue, is not always recognizable by inspection and palpation. An incision with a small pointed knife, a broader knife with the blade curved at the tip, a hypodermic needle, a trocar, and the like, may be found useful.

A *small knife*, even a needle or a probe, may be used to determine not only the nature but the extent of a swelling or a cavity. For instance, there is a soft or fluctuating swelling at the inner or upper-inner part of the orbit: an incision with a small knife will discover whether it is a hemorrhage, an abscess, a mucocele, a cyst, and what kind of cyst, simple, serous, sebaceous, or parasitic (echinococcus, etc.); a *probe* will *supplement* the diagnosis in exploring the walls of the cyst; further, in determining whether the orbital wall is intact, or carious, or perforated.

Of particular importance is the incision with a broader knife. Many tumors on the orbital walls may originate in the cellular tissue of the orbit, the periosteum, the bone, and the neighboring cavity. We may be in doubt whether it is an empyema, a sarcoma, or even an osteoma. In penetrating gradually deeper, by first dividing the integument, then the muscle, and so on, until the envelope or the substance of the tumor is plainly brought to view, we can make the diagnosis of the tumor and take the further steps accordingly. Whenever there is the least doubt I proceed in this way, being prepared for the entire operation, but advancing step by step, always making sure what part is before me. This is of the greatest importance in tumors behind the eyeball, in order to avoid cutting sensitive nerves, muscles, and the optic nerve, if they can be saved.

II. OPENING OF ABSCESSES.

After erysipelas, injuries, foreign bodies, operations on the eyeball, even on the muscles, acute diffuse or circumscribed suppuration may develop in the orbit. If the seat of an abscess is recognized by the tenderness of one particular place, by pointing and fluctuating, the knife is simply plunged into the abscess-cavity.

If the suppuration is diffuse, or if a number of small abscesses are scattered through the orbit, exploratory punctures in different places through the lids or conjunctiva are indicated. Even if no pus is evacuated,

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as in orbital thrombosis, the incisions are beneficial in unloading the tissue. In order to protect important structures—for instance, the tendon of the superior oblique or the membranous wall of the lacrymal sac—incisions on a grooved director are advisable.

III. EXTIRPATION OF TUMORS OF THE ORBITAL CAVITY.

(1) *Vascular tumors* occur in two varieties: (*a*) with *undefined or ill-defined* walls. These are met with particularly in the anterior portion of the orbit, and not infrequently together with palpebral angiomas, or a portion of the tumor is in the orbit, the other in the lids, both connected by one or several vascular cords or bands. They are almost all congenital, and increase slowly. To treat them with *ligation, injection of coagulating substances, electrolysis, galvano-causis, and the like, is very unsatisfactory.* The only way to cure them is by *extirpation.* This should be done out of the healthy surroundings, avoiding cutting into the tumor. If only the afferent and efferent vessels are divided, which is, of course, unavoidable, the bleeding will not be excessive, for they, like all other orbital vessels, are small and soon stop bleeding. To avoid cutting into the tumor, and also to diminish the bleeding during the operation, I have pressed a *horn plate through the conjunctival sac firmly against the bony wall of the orbit behind the tumor.* This has served me a good purpose in dealing with some vascular tumors in the inner part of the orbit which had pierced the lids and spread outside in the skin. I extirpated the orbital portion from its posterior limit to the inner surface of the lids, but did not attack the palpebral portion at all. There was no more hemorrhage than in the extirpation of a fibroma. I expected the palpebral portion to shrink after its afferent vessels, its supply-pipes, had been stopped, and so it did, gradually fading away. One of these patients, on whom I operated when she was a child of several months,[1] came to see me in April, 1897, twenty-two years later. She had grown a healthy woman, without any abnormity in her eyes.

In parenthesis I may say that I have operated successfully also in other parts of the skin, for instance, in the telangiectasias of the wings of the nose and adjacent parts of the cheek, according to the principle of destroying the afferent vessels by cauterization or ligation and leaving the rest alone. The vascular nævi and cavernomas then disappeared by shrinkage.

(*b*) *Circumscribed angiomas* are mostly met with as capsulated cavernomas in the muscle-funnel behind the eyeball. They are probably almost all congenital, compressing in their slow growth the surrounding connective tissue into the form of a white, firm, fibrous capsule. The interior is made up of erectile tissue. They neither pulsate nor cause increase of exophthalmus by stooping. Their differential diagnosis from a tumor of the

[1] See H. Knapp, On the Operative Treatment of Vascular Tumors of the Eyelids and the Anterior Segment of the Orbit, Archives of Ophthalmology and Otology, vol. v., 1876, pp. 514–524, and Archiv für Augenheilkunde und Ohrenheilkunde, Bd. vi. S. 38.

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optic nerve or a retrobulbar fibroma will rarely be possible before the extirpation. There is a small number of successfully operated retrobulbar cavernomas on record.[1] They can be removed from either the nasal or the temporal side. This should depend on the direction of the exophthalmus, the result of palpation, and the restriction of mobility, from which we may infer whether the bulk of the tumor is situated more to the nasal or to the temporal side. It seems that these tumors favor the nasal side and develop more towards the inner orbital wall.

Technic.—With the proper precautions and under general anæsthesia we open the conjunctiva vertically, suppose, on the inner side, between the caruncle and the cornea, penetrate deeper, turn the eyeball temporally, and now feel, which we did not before, the edge of a hard, fibrous mass behind the eyeball through the internal rectus muscle. If the tumor can be felt up and down, and the eyeball is much protruded, so as to let us assume with reasonable probability that we have to deal with a growth of considerable size, we detach the tendon of the internal rectus and pass two sutures through its proximal end, the needles of which we leave on the threads. With these threads we can draw the muscle towards the nose, while the eyeball is turned towards the temple with a sharp double-hook. We now work with a curved hand-chisel between sclera and muscle into the depth down to the mass of the tumor, which on exposure appears as a moderately firm mass with a smooth, somewhat bluish surface. This makes the diagnosis of a cavernoma probable. We work our way around the tumor, very carefully, through the surrounding connective tissue, using the scissors only when the chisel does not advance, and by very short strokes. This is particularly necessary along the posterior surface of the sclerotic. We cannot help cutting the ciliary nerves and vessels, but we want to spare the optic nerve, for the patient usually has still moderately good sight and only a low degree of choked disk. The optic nerve gradually can be felt on the outer side of the growth. We work along the latter from all sides, always holding the instruments pointed towards the surface of the tumor, never away from it. The growth becomes more and more movable; we can take hold of it with long and strong forceps, pull it gently forward, free its posterior surface, and pry the whole mass out with the curved hand-chisel. The tumor is clean, oval, moderately hard. We look at the pupil, and throw light on it; it moves, making it sure that the optic nerve has not been cut. After the insignificant bleeding, we stitch the tendon of the internal rectus to the sclerotic as in an ordinary advancement. The eyeball has its natural position, it moves in every direction, and there is no diplopia. We cleanse the eye and examine it with the ophthalmoscope. The condition is as before the operation, and the retinal arteries pulsate on pressure; we are sure that the central retinal vessels have not been injured. The

[1] See H. Knapp, A Cavernous Angioma in the Depth of the Orbit. Removal, with Preservation of the Eye. Archives of Ophthalmology, vol. xxv., 1896, p. 116.

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visual test gives vision and field as before the operation. The eye is bandaged, and the patient goes to bed; the other eye is also closed, except when he eats. The patient makes an undisturbed recovery, and leaves the hospital on the twelfth day. He returns in three months, perfectly well, the eye a shade deeper in the orbit than the other.

This is the kind of operation we should try to bring to a successful end. Yet we are not always so fortunate as to accomplish it. It may be possible to get the tumor out without detaching the muscle, particularly in case the tumor is to the temporal side, where the lower-outer portion of the orbit gives more space both to the palpating finger and the instruments. As soon as the tumor can be felt, the belly of the muscle is drawn up and the tumor extracted through the interval between the lower and external recti. On the other hand, it may not be impossible to extirpate the tumor without removing the eyeball. This was the case in a patient of Dr. S. C. Ayres, of Cincinnati, who suffered from the exceedingly rare tumor of a retrobulbar *lymphangioma*. Ayres's case is the second of this kind on record:[1] the first is described by Förster.[2] In its clinical features and the size and position of the tumor Dr. Ayres's case is identical with the case of hæmangioma which I described and depicted (*loc. cit.*, vol. xxv. of *Archives*), but his was a true lymphangioma. Dr. Ayres was kind enough to send me the specimen, and I could verify his diagnosis in every respect. (See the reference in my case, *loc. cit.*)

(2) *Fibroma.*—The pure, hard fibroma is a very rare tumor in the orbit. I shall in brief detail one case I had to operate recently. Exophthalmus with a secondary direction outward; inward mobility restricted; sight and fixation good. In the inner portion, deep in the orbit, a hardish tumor felt, immovable on the inner orbital wall; no pain on pressure; slow growth; congestion of optic disk; nose normal; probable (but erroneous) diagnosis, periosteal fibrosarcoma.

Operation.—Incision along the whole inner wall of the orbit, through skin and ligamentum canthi. Incision made deeper in an exploratory manner down to tumor. Hard, even fibrous edge. On detaching it from its surroundings it was found that it was not connected with the periosteum, but attached to it by a very slender layer of connective tissue, the same which surrounded the tumor on all sides and made it adhere to the lacrymal sac and Tenon's capsule, out of which it could be dissected and removed without wounding any important structure. The tumor was oval, fully two and a half centimetres in diameter and two centimetres in thickness; was hard, white, and like a scirrhus. The microscopic examination proved it to be a *chondrofibroma*. The wound was closed with sutures. The patient stayed in the hospital two days, the further treatment being carried on by his physician. The case exemplified the advisability of

[1] S. C. Ayres, American Journal of Ophthalmology, 1895, p. 21.
[2] Förster, Archiv für Ophthalmologie, Bd. xxiv., 2, 1878, S. 108.

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making the first part of any orbital operation in an exploratory manner, and then going on according to the conditions found. I prefer to proceed in this way, laying the tumor bare, and immediately removing it, instead of making an exploratory puncture with a small knife or a hypodermic needle, or trocar, then tell the patient and his relations all about it, and perform the operation later, if they still consent. If I am reasonably certain that an operation should be done, I prepare myself for all the eventualities that I can foresee, begin the operation, and proceed with it in an exploratory manner, until I see my way clear before me, but avoiding all dilatoriness as far as I can.

(3) *Tumors of the Optic Nerve.*—These tumors are not so rare as the fibromas and the retrobulbar angiomas, yet they are not of usual occurrence. Their diagnosis may long be difficult. Straightforward protrusion of the eye, congestion, optic neuritis, and freedom of movements are the chief symptoms. If the tumor can be felt, the diagnosis is easier. The smaller tumors present an oval swelling in the optic nerve,[1] the larger ones rest with a concave basis on the posterior part of the eye.[2]

If they are not very large, they can be removed with preservation of the eyeball. The first operation of that kind was performed by myself in 1874. The tumor, which I described as alveolar cancer, was in reality an endothelioma, a pseudoplasm which was not yet known at the time of my publication. I made an incision in the conjunctiva, laid the edge of the tumor bare, had the external rectus drawn up, isolated the tumor on all sides, cut off the central end of the optic nerve, detached the concave basis of the tumor from the posterior portion of the sclerotic, severed the optic nerve, drew the pear-shaped tumor out, and closed the wound with sutures. The healing was smooth. There were hemorrhages in the retina, followed by extensive atrophy of choroid and retina. The eyeball was in proper shape at first, but gradually shrunk, so that now (I saw the patient in November, 1896) it is completely shrivelled up. Yet the patient has had no complaint ever since the operation; she is well, and there is no symptom of a local or metastatic recurrence.

In the case of Dr. Gruening, where the tumor, a myxoma, was much smaller, the eyeball has kept its shape.

Lagrange[3] enlarges the external commissure, detaches the external rectus, frees the tumor from all its connections, draws it out, and stitches the muscle to the sclerotic again.

If the tumor is large and the eye totally blind, the removal of the latter will facilitate the operation very much. It might be possible, as Czerny[4]

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[1] Compare E. Gruening, Archives of Ophthalmology and Otology, vol. v., 1876, p. 508.

[2] Compare H. Knapp, *ibidem*, vol. iv., 1874, p. 323.

[3] Lagrange, De la conservation du globe de l'œil dans l'extirpation des tumeurs du nerf optique, Congrès français de Chirurgie, Paris, 1892, 18 à 23 Avril.

[4] Czerny, Klinische Monatsblätter für Augenheilkunde, Bd. xii. S. 447, in the discussion of a paper by Knapp, Bericht der ophthalmologischen Gesellschaft, Heidelberg, 1874.

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suggests, to remove a tumor from the optic nerve and preserve sight. The tumor may originate in the optic nerve sheath, invade only a part of it, and leave the optic nerve free.

Braunschweig[1] proposes temporarily to resect the outer orbital wall according to Krönlein, in order to get free access to the tumor.

The removal of tumors of the optic nerve is a beneficial operation, most of these pseudoplasms being benign, and not recurring or extending, even if portions of the optic nerve are left in the optic canal.[2] On the other hand, I dare say that some patients of mine who had the symptoms of optic nerve tumor, but refused the operation, have been observed for several years without much change in the exophthalmus and their general condition.

(4) *The resection of the optic nerve* (*neurectomy*) is not exactly an extirpation of a tumor, yet the method of operating is the same. The incision may be made on the nasal or on the temporal side. The nasal side has the advantage of a smaller distance to get at the nerve; the temporal side, however, offers a freer field and an easier way to divide the ciliary nerves. A meridional or crucial incision of the conjunctiva is made between the external and inferior recti, the external rectus drawn up with a hook, the cellular tissue divided to get access to the nerve, the eyeball drawn nasally with a sharp double-hook as far as possible, the nerve seized with curved forceps, pulled into the wound, and first cut as far centrally as the forceps can be advanced, then cut close to the sclerotic, and the piece between the two cuts pulled outward and exsected. It is, however, just as convenient first to sever the optic nerve at its entrance into the globe, then pull the piece forward with forceps and remove as much as is intended. The eyeball is rolled on its vertical axis until its posterior side is exposed. All the tissues on the posterior segment are then cleanly cut with curved scissors, especially the ciliary nerves and the blood-vessels.

It is somewhat easier to resect the nerve after first dividing the external rectus muscle, and stitch it together again when the optic nerve is removed and the posterior side of the sclerotic freed from all its attachments. The operation is sometimes followed by excessive hemorrhage, which in its turn may be followed by sloughing of the cornea from exposure, on account of the ensuing high degree of exophthalmus. It may be advisable to remove the eyeball at once if the protruding eye cannot be covered by the lids. Orbital cellulitis and panophthalmitis have been observed after neurectomy.

Indications.—This operation, as well as *neurotomy*, the mere division of the optic nerve close to the sclerotic, was extensively practised fifteen years ago to replace enucleation of the globe for preventing or curing sympa-

[1] Braunschweig, Die primären Geschwülste des Sehnerven, Archiv für Ophthalmologie, Bd. xxxix., 4, S. 59.

[2] Compare Salzmann, Studien über das Myxosarkom des Sehnerven, Archiv für Ophthalmologie, Bd. xxxix., 4, S. 94.

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thetic exophthalmia. It has gradually lost favor since several cases were reported in which sympathetic ophthalmia developed after neurectomy as well as after *neurotomy*. Enucleation is now considered the surer and safer operation. Neurotomy or neurectomy is still, but rarely, practised to remove neuralgia in blind but not disfigured eyes, or in stumps where prothesis is painful.

(5) *Serous, atheromatous, and dermoid cysts* are sometimes met with, especially in the anterior portion of the orbit. Their removal, according to the above-described rules of laying them bare, stratum by stratum, with the knife, a curved hand-chisel, and scissors, sometimes using a grooved director, can in many cases be done without opening the bag; in others, for instance the thin-walled echinococcus cysts, it is easier, and almost as sure, to open the bag, liberate its contents, and inject a strong antiseptic solution, such as tincture of iodine, nitrate of silver, and the like. We should not forget that these orbital cysts are sometimes multiple, as in a case of Cornwell.[1] We should, of course, endeavor to remove them without sacrificing the eye, and without producing diplopia.

(6) *Encephalocele* is a rare form of orbital tumor and difficult to deal with. It should be intrusted to such hands only as are skilled in the surgery of the head.

(7) *The lacrymal gland* in its two portions, the *orbital* and the *palpebro-conjunctival*, is not infrequently the subject of surgical interference. The *healthy gland has been removed* for troublesome and otherwise incurable *lacrymation;* if it is *degenerated* into an *adenoma*, an *adenosarcoma*, or a *cancer*, its extirpation is imperative. As with the parotid, its tumors are mostly mixed pseudoplasms.

Technic.—(*a*) *Extirpation of the Orbital Gland.*—Bearing in mind the precise position of the lacrymal gland in the upper-outer depression of the orbital wall, we shall have no difficulty in removing it. Under general anæsthesia an incision is made along the supero-external border of the orbit, deep to the bone, from the junction of the middle to the outer third of the upper border down to near the insertion of the outer canthal ligament. With retractors the lips of the wound are parted, the incision is continued to the surface of the gland, and the latter is shelled out of its niche with a curved hand-chisel or the closed ends of curved scissors. The exterior surface of the gland is first and easily freed from the conjunctiva, then the upper (or lower), then the external from the bone, and last the lower (or upper) from the conjunctiva. The posterior connection is likewise easily severed. If there is considerable hemorrhage, the lacrymal artery is ligated.

(*b*) *Extirpation of the Palpebral Lacrymal Gland.*—Under local anæsthesia the upper lid is drawn upward and off the eyeball. The patient

[1] Cornwell, A Compound Dermoid Cyst of the Orbit, Archives of Ophthalmology, vol. xi., 1882, p. 333, and Archiv für Augenheilkunde, Bd. xiv. S. 120.

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looking down, the exposed palpebral gland, about the size of a hazel-nut, is seized with toothed forceps, its conjunctival cover excised, and the glandular mass dissected, which, on account of the softness of the structure and its uneven surface, is not so easy as with the orbital gland. Yet, with care and perseverance, it can well be done without sacrificing the conjunctiva or leaving portions of the gland behind. The glands along the upper fornix, of which the palpebral gland is only an enlarged portion, are not interfered with. The wound may be left open or united with catgut or silk sutures; the lids are closed over them, and the eye is bandaged. The efferent ducts of the orbital gland are cut and closed by this operation.

Results.—In many cases the lacrymation is reduced to the normal standard, or so much lessened that the discomfort is almost entirely removed. This object is obtained frequently when the palpebral gland alone is removed; in other cases the extirpation of both glands of one eye, and in some the extirpation of the lacrymal glands on both sides, is required. Even in the latter case the secretion of tears will not be abolished as long as there is no atrophy of the conjunctiva, as in advanced cases of cicatricial trachoma,—xerophthalmus. This seems to indicate that the secretion of the lacrymal glands could be eliminated by closing up the tear-ducts. Bettremieux[1] has obliterated the excretory ducts with the galvano-cautery by repeated cauterization with the pointed electrode.

(8) *Pulsating exophthalmus* is mostly due to a *traumatic arterio-venous communication in the cavernous sinus.* The main operation is ligation of the common carotid, which cures one-sided and even double-sided pulsating exophthalmus.[2] This is easily understood if we bear in mind the communication of the two cavernous sinuses by the circular sinus of Ridley. In Dr. Gruening's case the blindness in both (protruded) eyes was cured at once by the ligation of one common carotid. Though this is an operation of general surgery and need not here be described, the ophthalmic surgeon should be prepared to perform it at any time in case of dangerous hemorrhage. The ligation of the carotid has to be done sometimes on both sides for pulsating exophthalmus in one eye, and even if done on both sides it is by no means a sure cure. In the majority of cases the patient finds much immediate relief, and the improvement holds on for many months, so that the patient considers himself permanently cured, but then gradually the bruit in the head, the exophthalmus, and the enlargement of the veins return, and another operation is necessary. In neglected cases the cornea sloughs from exposure, and when, after the temporary beneficial ligation of the common carotid, the veins begin to fill and the stump to protrude again, ligation of the carotid on the other, still unaffected eye is rather a

[1] Bettremieux, Traitement du larmoiement par la galvano-cautérisation des conduits excréteurs de la glande lacrymale à leur émergence dans le cul-de-sac conjonctival, Journal d'Oculistique du Nord de la France, 1893.

[2] See the remarkable case of Dr. E. Gruening, Archives of Ophthalmology and Otology, vol. v., 40, 1876, and Archiv für Augenheilkunde und Ohrenheilkunde, Bd. v., 1876, S. 280.

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hazardous undertaking. In one such case, where, at my request, the common carotid had been, for a time successfully, tied by the late Dr. H. B. Sands, in November, 1880, there was a relapse, the cornea sloughed, and the pulsating, very much swollen orbital tissue protruded so much that I *removed the contents of the orbit after enucleating the eye and ligating an enlarged pulsating blood-vessel*[1] *at the apex of the orbit, the superior ophthalmic vein.* When the orbital tissue was removed as a whole, the ligature was accidentally cut, yet there was no hemorrhage, probably because the stump of the vein was compressed by its retraction into the superior orbital fissure. "The calibre of the vessel when it was cut was six millimetres in diameter, its walls fully one millimetre in thickness. The orbital tissue removed formed a rounded tumor fifty-five by twenty-two millimetres." (*Loco citato*, p. 207.) The exenteration of the orbit was done two years after the ligation of the common carotid. The patient made a good recovery, and showed herself eight months after the operation, and later, with no recurrence. For fuller information on pulsating exophthalmus I refer the reader to the exhaustive monograph of H. Sattler in Graefe-Saemisch's Handbuch der gesammten Augenheilkunde, Bd. vi., 1880, Ss. 745–948.

(9) *Sarcoma.*—This kind of tumor rarely originates in the cellular tissue of the orbit, but mostly in the eyeball, upon the eyeball, at the orbital walls, and in the adjacent cavities. It requires extirpation out of the healthy surroundings. We may proceed in an exploratory manner, but soon we shall be unable to distinguish the healthy from the diseased structure. Sarcoma, like the other malignant growths, has no capsule; its elements invade the neighborhood without discernible lines of demarcation. Its consistence being different from, commonly greater than, that of its surroundings, we must take the sense of touch as our guide, introduce a well-sterilized finger into the wound, and cut the tumor out at some distance from its limits. The cells which invade the neighborhood imperceptibly penetrate also into the muscles, the conjunctiva, and the episcleral and glandular tissue. Even when we think that we have taken a good deal of sound tissue surrounding the tumor, its elements in a small number are already present in the adjacent structures, implicating them in their destructive growth, which will soon show itself as a local relapse.

According to diminished resistances, the tumor elements travel in different directions through the loose tissue of the orbit, producing that rapidly destructive form which is represented by the *multiple sarcoma* of the orbit.

All this shows that the *results of extirpation of orbital sarcoma are utterly unfavorable;* even if we enucleate the eyeball and remove the whole orbital contents, the periorbita included, the relapse mostly follows speedily upon the exenteration. It is questionable whether we prolong life by

[1] See H. Knapp, Relapse of Pulsating Exophthalmus cured by Extirpation of the Aneurismal Varix of the Orbit, Archives of Ophthalmology, vol. xii., No. 2, p. 221, 1883, and Archiv für Augenheilkunde, Bd. xiii. S. 375.

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extirpating a parvicellular orbital sarcoma; in most cases it looks as if the relapse spread farther and grew more rapidly than the original tumor would have done if left alone.

The *orbital sarcomas, therefore, offer only a very doubtful indication for extirpation.* There is one species of tumor which is clinically easily confounded with sarcoma, and which under the microscope presents the very picture of a small-celled sarcoma, but which is innocent and should not be operated on at all: I mean the *lymphoma* or *lymphadenoma.* The patients have a sallow complexion, are anæmic and lymphatic. We should examine their glands, their blood as to the number of white corpuscles, etc. If the patient has a manifest picture of Hodgkin's disease, we know what we have not to do and what we have to do, but also on the suspicion of the presence of lymphadenoma we may treat the patient with increasing doses of liquor potassii arsenitis, etc. In view of the unfavorable prognosis of the extirpation of orbital sarcoma, we are certainly justified in treating the patient tentatively. I have been consulted by patients who had orbital tumors which, showing under the microscope the picture of sarcoma, were removed with or without the eyeball. Tumors which appeared in the other orbit, with the same symptoms, where the operation was refused, disappeared spontaneously in the course of months. During the last years I have made the diagnosis of orbital lymphadenoma in several patients; they recovered completely under arsenic treatment.

Tumors of moderate malignity, fibrosarcoma and epithelioma, also those of great malignity, as gliomas and small-celled sarcomas, furnish a justifiable indication of the removal of all the contents of the socket of the eye.

IV. EXENTERATION OR EVISCERATION OF THE ORBIT.

Technic.—The outer commissure is enlarged to the orbital margin or farther, the lower lid is pulled down, a strong knife, under guidance of the left forefinger, is passed along the orbital border to incise the soft parts down to the bone, then the same is done at the outer and upper orbital borders, and last at the inner, where the region of the lacrymal sac may be left until later. During the progress of the operation the extent of the disease will show us whether we should make a *partial* or a *total removal,* the periosteum included, of the contents of the orbital cavity. If the disease does not involve the periosteum and the bone, we may with knife and scissors take all the soft parts out. The package of tissue crowded in the apex of the orbital pyramid—viz., muscles, nerves, vessels, and the optic nerve—can be divided with a sharp curette or with curved scissors, best with those of Warlomont, which at the same time cut and compress the tissue. At the inner wall we have to spare the lacrymal sac. In many cases the disease, say a malignant tumor, has encroached upon the orbital wall, periosteum, and bone. This has to be removed. In certain cases, say in multiple sarcoma or glioma-relapses of children, the removal must be radical,—namely, a

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Total exenteration, from the beginning. The enlargement of the palpebral fissure and the splitting of the soft parts, periosteum included, along the whole orbital margin, are done as described above. The periosteum is scraped off the bone with a raspatory, leaving the package at the apex to be cut, and exercising particular care in the region of the tear-sac. In case the disease extends to the optic canal, its wall and the adjacent surfaces should be scraped with a sharp spoon. The bleeding is sometimes copious, but can be stopped by tamponing the stiff-walled cavity with aseptic gauze.

Care should be taken in working at the upper and inner walls, also in the angle between the two orbital fissures, for in these localities the bone is so thin as to be easily perforated and crushed.

If islets of diseased tissue are left, especially after partial exenteration, they should be carefully removed with scalpels or sharp chisels.

When all the diseased parts are removed and the bleeding is stopped, the skin-wound at the outer canthus is united by sutures, the cavity filled with sterilized gauze, and the eye kept closed with a bandage for two or three days, even longer. As soon, however, as there is oozing or the least odor from the wound, the dressing should be changed daily or every other day until the wound has healed by granulation.

About twenty years ago some surgeons, especially the English, in order to destroy all vestiges of malignant disease, not only removed the periosteum, but papered the denuded wall with zinc-chloride paste smeared on linen. In a case where Lawson used this treatment for scirrhus, almost the whole osseous orbit exfoliated and was drawn out in one piece.[1]

The *indications*, as stated at the beginning, consist in the removal of malignant disease in desperate cases.

Results.—Though we cannot expect to cure such diseases, the relief from suffering, offensive discharge, and hideous deformity is often sufficient to warrant the operation. In some cases it may prolong life, in others it may shorten it. The operation, if correctly performed, under careful aseptic and antiseptic precautions, is not dangerous in itself.

V. THE TEMPORARY OSTEOPLASTIC RESECTION OF THE OUTER WALL OF THE ORBIT TO EXPOSE THE DEPTH OF THE ORBITAL CAVITY.

In 1886 W. Wagner[2] recommended the temporary resection of a wedge-shaped piece of the outer orbital wall for the removal of foreign bodies, tumors, etc., in the depth of the orbit.

In 1887 Krönlein[3] published a very instructive case of removal of a

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[1] Quoted from Sattler, in Arlt's Operations on the Eye, Graefe-Saemisch, Handbuch der gesammten Augenheilkunde, Bd. iii. S. 436, after Hulke, in Transactions of the Pathological Society of London, 1867, p. 233.

[2] W. Wagner, Die Behandlung der complicirten Schädelfracturen, Volkmann, Sammlung klinischer Vorträge, Nrs. 271 und 272, Leipzig (reprint), S. 86.

[3] Krönlein, Zur Pathologie und operativen Behandlung der Dermoidcysten der Orbita, Beiträge zur klinischen Chirurgie, Bd. iv., 1, Tübingen, 1887.

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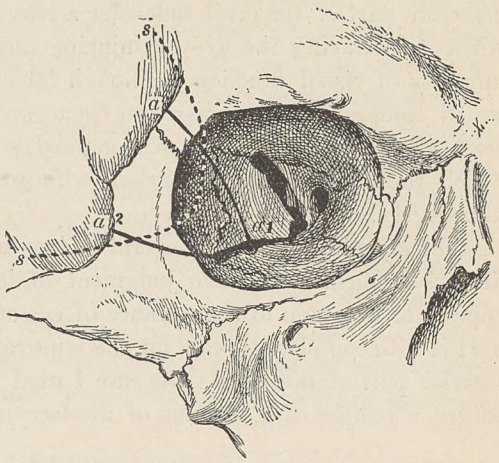
dumb-bell-shaped (*zwergsackartig*) dermoid cyst of the orbit by the above-mentioned operation, which since then has been repeatedly practised.

Technic.—A curved incision (see Fig. 47, *s*, *s*) is made from the temple over the upper-outer part of the orbital margin through the skin on the nasal side of the outer orbital margin, curving back into the temple above the zygomatic bone. The incision is carried down to the bone along the outer orbital margin. The periosteum on the inner surface of the outer orbital wall is detached with a raspatory as far as the anterior end of the lower orbital fissure, into which a sharp-pointed probe is inserted to serve as a landmark. A wedge-shaped piece (a, a^1, a^2) is now separated from the outer bony wall of the orbit by chiselling from the upper end of the margin obliquely down (a, a^1) to the inferior orbital fissure, and then horizontally from the lower end of the margin of the outer bony wall (a^2) to the anterior end of the inferior orbital fissure. The chiselling is not easy, for the bone is hard, readily splinters, and is thick and firm at the lower end of the outer orbital wall. A flat, sharp chisel should be used, working with gentle hammering into the bone with its corner, not with the whole sharp edge, which might splinter the bone. The piece of bone thus circumscribed is about three centimetres high and from three to four centimetres long. It remains in contact with all the soft parts on its edge and outer surface, which are subservient to its nutrition. If the periosteum which lines the inner surface of the outer wall is not diseased, it has only to be split to expose the posterior part of the orbital cavity, operations in which can then be performed with greater precision and more ease than if the outer orbital wall had been left in position.

FIG. 47.



Osteoplastic resection of outer orbital wall. (Krönlein's operation.)

When the operation on the contents of the orbit is completed and the hemorrhage stopped, the displaced bone is turned back into its place, and the periosteum united with catgut and the skin with silk sutures. A compressive bandage and rest secure immobility and smooth healing of the temporarily resected bone. It is useless to say that rigorous asepsis and neat operating are absolutely necessary, for the operative procedure is wearisome, readily leading to bruising of the periosteum and other soft parts, splintering of the bone, and infiltration of the surrounding tissue. I have done the operation once only, and would in future omit putting a sharp

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probe as a landmark into the occluding membrane of the lower orbital fissure. The probe can be dispensed with, and, without affording a sufficient compensation, it opens a door through which the wound-secretion penetrates into the spheno-maxillary fossa.

Krönlein's operation is *indicated:*

For the removal of tumors, foreign bodies, and all substances in the posterior part of the orbit that offer a reasonable chance of being removed without sacrificing the eye or injuring any part that is essential to the integrity of visual function. Though I have succeeded in removing deep-seated tumors and preserved the organ and function of the eye intact, I acknowledge the advantage of a method, so useful in general surgery, by which these operations can be done with greater ease and accuracy and the same safety.

The *experience* with this operation,[1] as far as I am informed, is still too limited to form a *de facto* judgment of its possibilities and merits. It appears to be a step in the advance of eye-surgery. I know that a surgeon used it with complete success for the removal of a small tumor in the outer-anterior part of the orbit; the case I used it in was unfavorable from its nature, a relapse of a sarcoma of the lacrymal gland.

VI. OPERATION FOR CARIES OF THE WALLS OF THE ORBIT.

The *walls of the orbit* are not rarely the *seat of caries*, which if left alone ends in more or less extensive destruction and exfoliation of necrosed bone and subsequent deformity from indrawn scars. It is mostly met with in children of scrofulous habitus, which in the majority of cases means *tuberculous disease.* The OPERATIVE TREATMENT consists in the *removal of all the carious and necrosed parts* with knife, chisel, and sharp spoon. *Blepharoplastic operations* are frequently required later.

VII. OPERATIONS FOR SARCOMAS OF THE ORBITAL WALLS AND THE NEIGHBORING CAVITIES.

These diseases are among the most dangerous that befall the visual organ. In their clinical and pathological aspects they are so much alike that their treatment should not be described in different places. Extirpation, as we have seen, is the operation for the sarcomas which develop in the orbit. Those on the orbital walls are mostly of periosteal or rather osteoperiosteal origin, frequently enclosing osseous spiculæ. They grow more or less rapidly, and terminate fatally by expansion and exhaustion

[1] Braunschweig, Die primären Geschwülste des Sehnerven, Archiv für Ophthalmologie, 1893, Bd. xxxix., 4, S. 1. Schreiber, Ueber osteoplastische Operationen der äusseren Orbitalwand, 11. Jahresbericht der Schreiber'schen Augenheilanstalt, Magdeburg, 1893, sowie Bericht über seine Thätigkeit in den Jahren 1895 u. 1896, Magdeburg, 1897, S. 22. W. Klingelhöfer, Eine durch temporäre Resection der äussern Orbitalwand extirpirte Orbitalcyste, Archiv für Augenheilkunde, Bd. xxxv. S. 86, and Archives of Ophthalmology, January, 1898, vol. xxvii. p. 25.

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rather than by metastases. There is no other treatment than *thorough and early removal*, but the prognosis is so bad that in many cases we may well abstain from any operative interference. This refers especially to the soft sarcomas of childhood, which after the most radical removal recur in a short time. In adults the prognosis is less gloomy, and there are exceptional cases in which, as in intra-ocular sarcomas, there is no recurrence, at least for many years. One case—a man of about thirty-three years—in my own practice presented a large sarcoma on the outer wall which had caused considerable exophthalmus and encroached upon the external rectus muscle. I removed it radically, taking away the periosteum, scraped the bone thoroughly, cut off all soft tissue in the outer half of the orbit, including the external rectus, denuding the outer half of the sclerotic. The recovery was undisturbed; the eyeball was immovably united to the outer wall, but when last seen, which was about ten years after the operation, it had not lost its shape. The microscopic examination showed the structure of a sarcoma. The case is so exceptional in my experience that I am inclined to believe that the tumor originated in the lacrymal gland, the tumors of which afford a better prognosis than the sarcomas developing in the tissues or the walls of the orbit.

Still worse are the sarcomas originating in the *neighboring* cavities and extending into the orbit. Before they encroach upon the orbit they have more or less filled the adjacent sinuses, so that we find them in the ethmoidal, frontal, and sphenoidal cavities, and in the nasal passages and the naso-pharyngeal space. I have once been requested, and was obliged to consent, to operate in such a case on which ten previous operations had been made. The growth filled the orbital cavity, the nasal passages, and the retropharyngeal space so extensively that the patient had great difficulty in breathing, and was in danger of suffocation. I removed all the obstruction, cleansing the walls of the cavities with a sharp spoon. There was no dangerous hemorrhage, and the patient lived thirteen months longer. He was a man of sixty years of age.

Still more distressing are, as I have said, the *sarcomas of the neighboring sinuses in children*. They cause exophthalmus with such rapidity that on seeing it for the first time we naturally think of an inflammatory, at least circulatory, affection in the orbit. Yet there is no pain, no fever worth speaking of, and no hardness or circumscribed resistance felt anywhere. In a few days, however, or somewhat longer, while the protrusion of the globe has progressed, a distinct swelling is felt in the depth of the orbit on the nasal side. I can explain this sudden appearance and rapid growth of orbital sarcomas only by supposing that they develop in one of the neighboring sinuses, especially the ethmoidal, fill them completely, are compressed by the unyielding bony walls, and when these are perforated the compressed tumor-masses invade the loose orbital tissue and expand in it with astonishing rapidity.

A girl of two years who presented these symptoms typically was oper-

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ated on by me three years ago. I beg to report her case, as it exemplifies the whole group not only in its clinical features, but also in the *manner and extent of the operations possible in such cases.* The child had no distress in breathing, and no particular obstruction of either nostril was discovered. The exophthalmus was on the left side. As soon as I felt the tumor I advised the operation, and performed it the next day. I proceeded in an exploratory way, thinking that an empyema might be present. Soon the border of the fleshy substance was reached, and the pseudoplasm removed with scissors and the orbital wall scraped with a sharp spoon. There was no noticeable defect in the wall. Recovery was rapid.

A local relapse, which in about two months had made its appearance and pushed the eyeball forward, was somewhat later, during my absence in Europe, operated on with removal of the eyeball by my associate, Dr. R. O. Born. This was very soon followed by another relapse, and, as the very intelligent and reasonable parents were informed of the incurability of the disease by further operating, Dr. Coley was asked to try the treatment with *injections of the toxine of erysipelas.* During the first weeks this treatment seemed to stop the growth of the pseudoplasm, but it soon proved to be a vain hope.

When I returned from Europe and saw the child again, the orbit was nearly filled, and also the left nostril and left choana were obstructed. Being asked for my opinion, I had to declare to the parents that I considered the disease of their child incurable by any means now known. They said, "What will become of the child if not operated on again?"—"'The tumor will continue growing, slough on the surface, but extend in the depth.'"—"Will the child be disfigured and suffer much?"—"'These tumors in their progress present a saddening aspect and emit a nauseating secretion. They cause death by exhaustion of the system or involvement of an important organ.'"—"What will be the result if you operate on the child again?"—"'If I remove the malignant growth as radically as surgical art permits, the child will not suffer from the wound, but the disease will continue its progress into the skull, and the child will die from compression of the brain.'"—"Under great suffering?"—"'Probably with no suffering at all.'"

After a short consultation in a neighboring room, the father returned, saying, "If the child cannot be saved, we have concluded to spare it all the suffering we can. We request you to operate on it."

Under ether-narcosis I removed the growth from the orbit and adjacent cavities and carefully chiselled away the adjacent bone. The orbital cavity was connected not only with the naso-pharyngeal space and the sphenoidal sinus, but also with the cranial cavity. The bones of the apex of the orbit were chiselled and scraped away. Towards the end of the operation the cavernous sinus was freely exposed. It had healthy walls, was blue, pulsated, and felt soft. The wound was tamponed with aseptic gauze, as in radical operations in mastoid empyema complicated with epidural abscess. There was scarcely any reaction after the operation. Temperature, 100° to 101° F. The child was conscious, ate and slept well. On the twelfth day she became soporose, and in twenty-four hours passed away without a struggle. Autopsy was not permitted.

VIII. OPERATIONS FOR OSSEOUS GROWTHS IN THE ORBIT AND NEIGHBORING CAVITIES.

The *exostoses starting from the internal surface of the orbit* are mostly round and nodular, covered with periosteum, and give no trouble other than by compression and displacement of the eyeball and other structures,

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especially the optic nerve. They are mostly of very slow growth, may become stationary, are of rare occurrence, and seldom of large size. If they give trouble they should be removed. The *method* is that of chiselling them off from their base, not attacking them from the surface or chiselling or sawing them off piecemeal. The drills and saws moved by dental engines or electro-motors are no more expedient than the chisel. At the base of these growths the supporting bone from which they spring is, as a rule, somewhat cancellous and only of moderate density, yielding well to the chisel; whereas the substance of the growth is most compact, the growth eburneous, hard like a billiard-ball. If the growth is small, the operation may be begun by incising and stripping off the periosteum with a raspatory. If the growth is larger, a part of the periosteum at the crest may be left, but the remainder should be stripped back with a raspatory so as to free the base. The chiselling should be done with a flat chisel with light strokes, and so that only the corner of the chisel attacks the growth,—all this to prevent splintering of the orbital wall. There is no hemorrhage to be feared, and the healing is mostly without reaction, even if a neighboring cavity should have been opened. A considerable number of successful operations of this kind are on record.

This is not the case with the *exostoses of the neighboring cavities, of which the frontal sinuses furnish the largest contingent.* They may have a small pedicle, which can easily be severed from the base, and it is not very rare that separation from its base occurs spontaneously, leaving the tumor loose in the cavity. Others are sessile,—*i.e.*, adhere with a broad base to the wall, or in the frontal sinuses to the bony septum between the right and the left sinus. They are nodular, covered with mostly thickened periosteum, grow slowly, but up to a certain point steadily, perforate the adjacent walls, the orbital, the glabella, and the posterior wall, projecting as large tumors into the anterior fossa of the brain. Their differential diagnosis is not always easy. They project in the inner-upper corner of the orbit, feel hard, yet no more than certain empyemas which expand the orbital wall of the sinus. Even the harder sarcomas may present the same resistance and the same nodular surface on palpation. The osteomas occur in two varieties, the one with a thin shell and cancellous interior, the other compact, ivory-like, throughout. The neighboring bone is thickened in some cases (illustrated with a good drawing by Panas, Traité, p. 420), in others it is corroded and thinned. Besides the local symptoms, there is not infrequently headache, depression, or irritability, or we find outbreaks of severe cerebral disorder, occurring in acute attacks, which in a case of mine were diagnosed as typhoid. These symptoms, in the presence of a tumor at the roof or inner-upper corner of the orbit, are, it seems, a sure sign of penetration into the cranial cavity. Thirty years ago, Paget and others considered those tumors as beyond the reach of art, and even Berlin[1] found the fatal termination of operative

[1] R. Berlin, Graefe-Saemisch, Handbuch der gesammten Augenheilkunde, Bd. vi. S. 726.

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attempts so frequent that he declared these growths to be a *noli me tangere.* If left alone they may remain stationary, or may end fatally by meningitis, the germs entering from the sinus into the cranial cavity. This and the difficulties of an operation are illustrated by a case of mine published in 1861.[1] After the periosteum was detached an ivory-like tumor was found protruding over the inner-upper wall of the orbit. The *method of operating* may be exemplified by the following three cases.

(1) *Use of Saw and Chisel* (author's case).—"I *sawed with Heine's osteotome a furrow* into the ivory mass along the upper margin of the brow, and chiselled the part in front of it away thirty millimetres in length, eighteen in thickness. This was only a partial removal. The patient was too much exhausted to continue the operation. Moderate suppuration followed. The patient did well seven weeks, then meningitis set in, with intense headache. In the eleventh week after the operation he died. The autopsy revealed a large tumor starting from the frontal sinus; two-thirds of its bulk was situated in the anterior cranial fossa.—Meningitis."

(2) *Cancellous Bone with a Hard Shell* (Dolbeau's case[2]).—*Breaking and Removing of the Bony Shell with Forceps.*—After the tumor had been laid bare by a vertical incision along the nose and a horizontal one along the brow, Dolbeau seized it with forceps, and, as a piece of it broke off, he removed the remainder piecemeal, the tumor having a hard shell and a cancellous interior. It was connected with the septum, which in a small place showed a rough surface, its base. In smoothing this place with a rongeur, an artery spouted, and had to be obliterated with a globule of wax. The patient had a good deal of reaction. Swelling in the region of the wound on the fifth day, suppuration, headache, and some delirium, pallor of face, somnolence, pulse sank from 74 to 48. From the ninth day gradual improvement. On the thirty-third day discharged cured.

(3) *Subperiosteal Enucleation.*—Successful case of present writer.[3] Man of forty-eight; healthy. Tumor in upper-inner corner, of bony feel; increasing. Curved incision from trochlea along orbital margin and nose to ligamentum canthi internum. Periosteum carefully detached from nodular surface of bone. With continuous but gentle chiselling the tumor is detached from its bony surroundings all around and always under the periosteum, which is drawn aside with blunt hooks. After an hour's work the tumor became loose, and might have easily been drawn or pried out. This not being considered safe, for fear of lacerating the dura mater, the tumor was held firmly with bone-forceps and rotated gently on its vertical axis until the posterior surface was turned into the nasal cavity and the perios-

[1] H. Knapp, Beschreibung eines Falles von elfenbeinerner Orbitaexostose, Archiv für Ophthalmologie, Bd. viii., 1, 1861, S. 239.

[2] Dolbeau, Mémoire sur les exostoses du sinus frontal, Paris, 1871.

[3] H. Knapp, Subperiosteal Enucleation of an Ivory Exostosis of the Frontal Sinus, extending into the Nasal and Orbital Cavities. Healing by First Intention. Archives of Ophthalmology, 1880, vol. ix. p. 464.

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teum stripped off with a hand-chisel from the posterior and upper sides. When this was finished, the tumor lay entirely loose in its capsule and was removed without cutting a fibre. The wound was closed with sutures, only in the lower part a perforated silver tube was introduced for drainage. The wound healed by first intention. The patient felt somewhat weak, but was well in two weeks, and has remained so these seventeen years. He is an active man. The operation has left no trace. The dimensions of the tumor were $36 \times 29 \times 24$ millimetres.

THE OPERATIONS FOR EMPYEMA OF THE NEIGHBORING SINUSES OF THE ORBIT.

I. The empyema of the *frontal sinus* is the best known and for the eye surgeon the most frequent of the affections of the neighboring sinuses. I shall, therefore, treat of it first.

The *first stage of the disease is commonly occult.* It is the propagation of catarrhal rhinitis into the sinus. The fronto-nasal canal is narrowed by swelling of its walls, leading at first to intermittent, later to permanent retention of secretion in the sinus. The symptoms are those of chronic or subacute rhinitis with attacks of frontal headache. This stage rarely is brought before the ophthalmologist, but is amenable to treatment, both by local application of antiseptics and caustics to the mucous membrane of the middle nasal meatus and by the *removal or destruction of polypoid granulations at the infundibulum and the adjacent mucous membrane.* Rhinologists at present are fully awake to the importance of treating just this region of the nasal passages.

The *second stage*, a *swelling along the upper border of the orbit, mostly in the inner-upper corner*, is seen and treated by ophthalmic surgeons. It commonly presents a hemispherical elevation, of even surface and moderate resistance, over the surrounding orbital wall. Usually there is no tenderness or redness present, and fluctuation in many cases is not to be made out on account of the tightness of the wall of the tumor. The size of the swelling varies greatly, from one centimetre in breadth to the whole length of the upper orbital border. Its posterior limit cannot be ascertained. The eyeball is pushed forward and outward. The development of the empyema is slow, and even large tumors with pronounced exophthalmus may be borne for years without annoying the patient further than giving him attacks of frontal headache. Empyema may be mistaken for fibrosarcoma, and even for osteoma. Different operations have been made,—viz.:

(1) *Evacuation of the glairy, ropy, mucoid contents by simple puncture.* It relieves the patient for a time, but the bag will almost invariably refill.

(2) *Broad opening of the anterior wall, exploring the condition of the cavity with a probe, removing polypi (which are not rare) and scraping uneven or carious places with a sharp spoon, draining the cavity with a perforated silver tube or the usual sterilized elastic tubes, syringing the cavity with caustics (nitrate of silver) and antiseptics.* By this method a number of cases are

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cured,—it is stated about fifty per cent.; the others leave fistulæ. I have used this procedure for thirty years. In some cases there has been a speedy and permanent recovery; one patient, with purulent contents and extensive polypi, whom I have seen occasionally now for twenty years, has never had any trouble since the operation; others appeared cured for a few years, then they relapsed and had to be operated on again; others have worn their tube for years. They have to use a gold tube, which does not discolor the skin; they hide the flange with a small piece of isinglass plaster. They syringe the small fistula once every day or every other day, but have no other discomfort. A small quantity of the glairy, ropy secretion is always brought out.

In the *third stage, caries with fistulous openings.*

(3) *A radical operation has to be performed*, in the same way as in *chronic* suppuration of the middle ear, especially the attic and mastoid. *All the carious and necrosed parts have to be removed and the communication with the nose re-established or the cavity closed by granulation and epidermization.*

This treatment, in some form or other now in great favor, is not new. It was described in English and Continental literature, with reports of cases, more than one hundred and fifty years ago.[1] Panas in his treatise advocates it warmly (vol. ii. p. 474, 1894). He recommends trephining of the frontal or orbital wall, combined with a drainage-tube, which he draws with a curved catheter into the fronto-nasal canal. The catheter is introduced from the sinus through the fronto-nasal canal and the infundibulum into and out of the nose. At the end of the catheter is an eyelet through which a thread is passed and attached to an india-rubber drainage-tube. The catheter in being drawn back carries the drainage-tube along. One end of the tube comes out through the trephine opening, the other through the nose. The ends of the tube are tied together with a thread, which holds the tube in position. The frontal trephining has the advantage of direct inspection and easy treatment of the whole field, in removing polypi or carious bone, and scraping the fronto-nasal canal and any part of the sinus that shows disease. Through the drainage-tube antiseptic and alterative (modifying) liquids are injected. Panas says that some people have blamed the method for leaving a deformity. The cicatrix can be made inconspicuous if, in following the rule which he has laid down, we form a triangular flap consisting of the whole integument of the glabella and the periosteum, the base being above, one line along the median line, the other in the upper orbito-palpebral groove. Another objection of more weight, Panas says (*loco citato*, p. 475), is the length of time the treatment requires,—namely, months, even a year. To avoid this, the whole anterior wall of the sinus could be removed at the price of an ungraceful depres-

[1] L. H. Runge, Halleri Disput Chir., 1750, describes the obliteration of the frontal sinus by his father.

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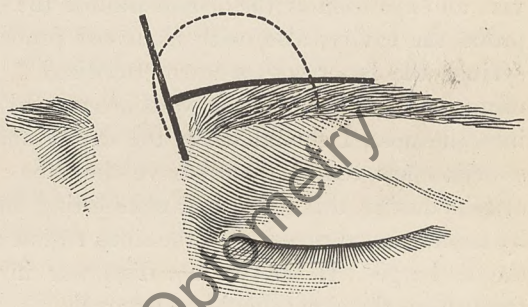
sion, as Kalt has done in one case, where the recovery was rapid. I have seen the result of such an operation done in New York. It left a deep and extensive, most disfiguring depression.

Kuhnt, in his exhaustive monograph on the "Inflammatory Diseases of the Frontal Sinuses," Wiesbaden, 1895, recommends essentially the same method as Panas. He told me lately that the disfigurement was avoided if the periosteum was preserved. Besides Kuhnt, the radical operation—obliteration of the sinus—is chiefly recommended by Nebinger[1] (from the anterior wall), and Jansen[2] (from the lower wall).

At the last International Medical Congress, held at Moscow, August 19–26, 1897, Dr. S. Golovine, of Moscow, read a paper on

(4) The *osteoplastic opening of the frontal sinus* according to Professor Czerny, which he had done in five cases with good results. Czerny had proposed it in 1895, after having tried it in one operation. Golovine said "that by experiments on the dead body he had succeeded in simplifying Czerny's method considerably. He demonstrated the method on a model. According to the 'Aperçu de l'activité du cercle ophthalmologique de Moscou depuis 1887–1897,' p. 95, he makes an incision along the upper border of the eyebrow (Fig. 48) from the nasal end to the centre, then another smaller one through the nasal end of the first incision from the lower margin of the brow obliquely upward and inward. A curved incision of about two centimetres in height is now made through the periosteum, the basis of which is formed by the inner third of the upper margin of the orbit. Along this line the bone is opened with a chisel. The osteo-periosteal flap thus obtained, and forming the anterior wall of the frontal sinus, can be easily lifted and turned on its base, the periosteum and soft parts of it remaining intact. Through this window the frontal sinus is now examined and scraped, and drainage into the nasal cavity by an elastic catheter is established. After this the flap is put back in its place and the skin-wound hermetically sutured." There has been primary union and a very satisfactory result in all cases.

Fig. 48.



This method, Dr. Golovine says, will induce us to undertake the operations on the frontal sinuses with more confidence, and also to make them for exploratory purposes in doubtful cases. (A detailed paper by Golovine will be found in the March, 1898, number of the *Archives of Ophthalmology*.)

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[1] Described by Praun, Inaugural Dissertation, Erlangen, 1890.

[2] Jansen, Archiv für Laryngologie, Bd. i. Nr. 2.

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If both sinuses are diseased, which is rare, Montaz has proposed to open both by trephining the glabella in the median plane. Panas thinks it will be sufficient to treat the less affected sinus by alterative injections from the other one, eventually after perforating the septum with a trocar.

INDICATIONS AND RESULTS OF THE ABOVE METHODS.

The *first stage* of frontal-sinus inflammation should be treated by a surgeon conversant with rhinological methods of investigation. Anterior rhinoscopy and probing the sinus through the infundibulum and fronto-nasal canal should precede any further treatment. In injecting liquids into the upper part of the nose, even with the intention of acting chiefly on the naso-frontal canal, we should be careful not to throw up a larger quantity of liquid, for this in penetrating into the sinus commonly produces a good deal of frontal pain. Disease-germs may in this way also be thrown into the sinus and produce inflammation. Applications with an atomizer are preferable to any kind of syringe. Cauterization, curetting, and, if polypi are present, snaring them off, will be necessary.

In the *second stage*, when there is nothing but a tumor in the upper region of the orbit, I would advise careful incision, to lay the wall of the tumor bare, see if it is purely membranous, or membranous and bony,—viz., an expansion of the orbital wall of the sinus,—make a small incision, probe the cavity, also, with a curved probe, the frontal canal, or try to syringe the latter with a warm sterilized 7 : 1000 salt solution by means of an Anel syringe. If the walls are normal and the probe or water passes into the nose, I should drain the cavity and syringe the canal until all secretion has ceased, then remove the tube. There may be inflammatory attacks during this treatment, manifesting themselves by frontal headache, by tenderness on pressure on the sinus region, and frequently by stoppage of the discharge. If they do not disappear in a few days, or if they return frequently, the sinus should be exposed.

In *relapses and fistulas*, the fistula and the interior of the sinus should be exposed and the parts treated by a method as radical and extensive as the open inspection and examination make it appear necessary. A broad opening of the orbital wall of the sinus, without injuring the superior oblique muscle and without cutting skin and periosteum away, affords in the majority of cases, as far as my experience goes, sufficient access to the diseased parts.

In *caries and necrosis*, especially when the fistula and the abscess are farther away from the inner corner, *temporary or permanent resection* of the anterior or anterior and lower walls will be the best treatment. If such a focus of dead and decaying bone with insufficient outlet of the secretion is left, dangerous and fatal complications may ensue,—namely, erysipelas, meningitis, and cerebral abscess. A swelling at the outer third of the orbital margin always awakens the suspicion that the sinuitis has been of long duration. Cerebral abscess from this cause may have existed for years

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without other subjective symptoms than heaviness and pain in the frontal region and attacks of fretfulness and depression.

The *results* of the operations for empyema of the frontal sinuses are gratifying. The local and cerebral symptoms disappear, and the eye returns to its position uninjured in structure and function. Without a radical operation a fistula may remain for years or a lifetime and a cerebral complication occur at any time.

II. *Mucocele and empyema of the ethmoid cells* encroaching on the orbit are not so frequent as those of the frontal sinus. Yet a number of cases have been described, among them six by myself.[1] They result from chronic catarrhal and purulent rhinitis, perforate the os planum, and form a subperiosteal, hemispherical tumor on the inner orbital wall. If they are confined to the ethmoidal cells, it is not dangerous to open the abscess from the orbit, explore the bone and the ethmoidal cells, remove carious and necrosed bone, as well as polypi, establish a communication with the nasal passages, and then stitch up the opening in the orbit. Bandaging and rest for a few days will cure the orbital complication. The nasal disease will require a longer treatment by antiseptic sprays, cauterization of diseased mucous membrane, and snaring off of polypi.

Empyema of the ethmoid is frequently associated with empyema of the sphenoidal and frontal sinuses, and then is not so readily cured.

III. *Empyema of the sphenoidal sinuses* is a disease which is not yet sufficiently appreciated as a cause of serious eye-disease. The proximity of the sphenoidal sinus to the optic nerves, from which it is separated only by a very thin bone, is the cause of optic neuritis and more or less rapid one-sided or double-sided blindness. Perforation of the bony wall of the sphenoidal sinus causes orbital swelling and exophthalmus, the same as the sphenoidal and ethmoidal empyemas do. The perforation is, however, more difficult to diagnosticate, for it occupies the tip of the orbital cone and cannot be reached by palpation. The exophthalmus is straightforward. Perforation of the abscess into the cranium causes death by meningitis. The diagnosis from the subjective symptoms and from a discharge into the naso-pharynx can be made certain on supplementing it by probing and *perforation of the anterior wall of the sinus*, which at the same time is the *proper operation* for the difficulty. A very instructive paper on this disease, with the report of three cases, among which was one complicated with blindness, successfully operated on, by C. R. Holmes, of Cincinnati, is published in the *Archives of Ophthalmology*, vol. xxv., 1896. The successful case was one of "purulent inflammation of the left sphenoidal sinus. Intense headache. Total loss of sight in the left eye. Opening the cavity. Recovery of vision." To get access to the sinus the middle turbinated had to be removed. The field was so masked with blood that the drill was directed more by measurement than by sight. It opened the cavity readily, and the improvement began at once.

[1] H. Knapp, Archives of Otology, vol. xxii., 1893, No. 3.

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IV. The *malar antrum* not infrequently causes eye-disease by *caries*, *empyema*, and *malignant tumors* which extend into the orbit. The *operations* for these affections need not be detailed here. They belong to the domain of rhinology and general surgery. Yet if the ocular complication is the most important feature of the disease, the oculist, in case he cannot have the assistance of a surgeon or rhinologist, should be prepared to do those operations himself. They do not endanger life, but, as in the case of empyema, may require longer after-treatment. The diagnosis is more difficult than the operation.

V. *Multiple Empyema.*—In most cases empyema is not confined to one sinus. The frontal-sinus empyema is mostly combined with empyema of the anterior ethmoidal cells, and very frequently with that of the antrum of Highmore. The orifices of those cavities are situated so near one another that the secretion from one cavity readily flows into the other cavities. The sphenoidal-sinus empyema is mostly combined with empyema of the posterior ethmoidal cells. In a few cases I have seen empyema in all the accessory sinuses of the face, with atrophy of both optic nerves, the disease having been recognized too late. I may mention that empyema may be the consequence of tumors, not only of polypi, which, like the empyema, may have a common cause, but also of sarcoma, and the true nature of the growth may not be apparent even on a macroscopic post-mortem examination, as empyema of the sinuses—for instance, of the sphenoidal—may produce so much thickening of the dura mater as to simulate a tumor.

§ XI. REMOVAL OF FOREIGN BODIES FROM THE EYE.

I. In the *conjunctival sac* foreign bodies of all descriptions are found. Their place of predilection is the shallow depression one to two millimetres behind the margin of the upper lid. They almost all lie there loosely adherent to the mucous membrane, and are easily *wiped off with cotton or a handkerchief*. In the bulbar conjunctiva they are often somewhat embedded, so that they cannot be wiped off, but have to be *dug out with a spud* (foreign-body needle), as also at times in the palpebral portion. When they are difficult to get out with a spud, they may without hesitation be *cut out*, seizing the implicated small piece of conjunctiva with fine forceps and cutting it off, together with the foreign body. In rare cases elongated foreign bodies with ragged edges, such as blades of grass or spikes of corn, work up under the upper lid and remain in the upper fornix for months. They cause some uneasiness, slight lacrymation and discharge, but no notable annoyance, until some swelling and tenderness of the upper lid prompt the patient to consult a physician. When we pass the finger over the outside of the upper lid, we feel a distinct though ill-defined tumor between brow and tarsus. Turning the lid, letting the patient look down, and pressing the fornix portion down with Daviel's spoon, we find the conjunctiva swollen and the foreign body more or less hidden in granu-

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lation-tissue and puriform secretion. *Removing the foreign body with the spoon or anatomical forceps and washing the conjunctiva out,* so that nothing of the foreign body is left, is all that it is necessary to do. The granulations will disappear of themselves. In examining for foreign bodies we always should think of the upper cul-de-sac, and evert it fully by pressing the fornix down, and also pass a Daviel spoon through it, so as to be sure that nothing remains hidden.

II. *Foreign bodies in the cornea,* if they are superficial, can often be *wiped off with absorbent cotton* wound on a probe or match, or, if this is insufficient, by a *quick stroke with the sharp edge of a foreign-body needle.* If this will not answer, *the cornea should be anæsthetized with cocaine* (or rather holocaine, the action of which is quick and transient), *steadied with the finger or fixing-forceps, and the foreign body well illuminated and dug out with a needle or a small gouge.* If there is a ring of gray infiltration around its seat, or a small brown or black disk, in case the foreign body consisted of iron, it is better to scrape that off too. A handy instrument is a needle on one end and a gouge on the other, both movable in an ivory handle, so that they are both covered when not used, and the one or the other is out when used.

FIG. 49.

Spud and gouge to remove foreign bodies from the cornea.

It is very necessary that we use proper antiseptic precautions in removing foreign bodies from the cornea; also that we sterilize the wound, if it is infected, with tincture of iodine, argenti nitras, etc. If the foreign body is *pointed and has penetrated more deeply into the substance of the cornea,* we should with a cataract-knife slit the overlying lamellæ open, and dig the foreign body out from the side or from behind forward, always being careful lest we should by injudicious scraping push it deeper.

If, as may happen in rare cases, an elongated, sharp-pointed foreign body has entered the cornea so deeply that its tip protrudes into the anterior chamber, whereas the body is held fast in the cornea, the safest way to get it out is first to pass a broad needle or a Beer's knife behind it into the anterior chamber and try to press the body backward. In case this attempt is unsuccessful, we hold the blade behind the foreign body in order to prevent it from falling into the anterior chamber during the act of digging it out. This operation is not so hazardous as it would seem to be.

In a case where a triangular small stone[1] was held with its thicker portion in the cornea, whereas the point stuck out into the anterior chamber, I made a corneal incision above the point of the foreign body. Trying in

[1] H. Knapp, Removal of Foreign Bodies from the Interior of the Eye, Archives of Ophthalmology and Otology., vol. vii. pp. 307–346, and Archiv fur Augen- und Ohrenheilkunde, Bd. viii. S. 73.

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vain to draw the foreign body out through the anterior chamber, I seized the point with a pair of anatomical forceps, and pushed it backward until the base could be grasped with another pair of forceps, by which the whole foreign body was removed. Recovery.

III. *Foreign bodies in the anterior chamber can be removed with a grooved hook.* (Fig. 50.) An incision being made with a lance or a knife, the hook should be passed behind the foreign body and the latter drawn out.

FIG. 50.

Grooved hook to remove foreign bodies from the interior of the eye. (Knapp.)

If the foreign body is attached to the iris, a flap incision should open the anterior chamber, the flap should be raised and the foreign body picked up. If this procedure is not successful, the piece of iris holding the foreign body should be made to prolapse, so that the foreign body can be directly seized and detached from the iris, or, if this is not possible, the piece of iris should be cut out, together with the foreign body. In the former case the iris is reduced. Several successful operations of this kind are on record, and I have had some in my own practice.

If the foreign bodies are of iron or steel, they may be extracted with the magnet, of which later.

IV. *Foreign bodies in the lens*, if the eye is quiet, should be left until the cataract is fully developed, and then be removed together with the lens by flap extraction. If the capsule is extensively torn and the eye irritated, as much of the lens may be let out through a corneal curved incision as will escape without prolonged manipulation; if the foreign body does not accompany the escaping part of the lens, it may be advisable to try to draw it out with the grooved hook, or, if of iron, with a magnet.

V. *Foreign bodies in the vitreous chamber* are difficult to deal with. They should be divided into two groups:

(*a*) *Such as are not of iron or steel.* If they float in the vitreous and can be seen, they can, as far as I have experienced, best be removed by making a meridional incision through the sclerotic near the foreign body, introducing the grooved hook, passing it, under the guidance of the ophthalmoscope,—in this case a forehead mirror,—behind the foreign body, and drawing the latter out through the opening. The foreign body easily escapes, but with patience and delicate handling we can succeed. I have had a few successful cases, especially with gun-caps, in my own practice.[1] The number of successful cases on record is, however, very small.

If the foreign body cannot be seen, we can scarcely think of extracting it, as the diagnostic symptoms—local pain, defect in the field of vision,

[1] H. Knapp, Transactions of the American Ophthalmological Society, 1873, p. 108, and Archives of Ophthalmology and Otology, vol. vii., 1877, p. 337, etc.

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circumscribed whitish-yellow opacity in the vitreous—will hardly ever be distinct enough to base on them the plan of an operation. Beginning panophthalmitis indicates evisceration of the globe or enucleation; insidious plastic or sero-plastic inflammation, especially irido-cyclitis, enucleation.

In some parts of Europe, especially Northern Germany, a living foreign body, *cysticercus cellulosæ*, is a frequent inhabitant of the eye, and is often —it seems mostly—successfully extracted. If it is subretinal, a meridional section of ten millimetres is made through the conjunctiva and Tenon's capsule, between two recti muscles, the wound held open with two small hooks, and the sclerotic divided with a cataract-knife, beginning three millimetres behind the cornea and passing from five to six millimetres backward. The scleral wound in its turn is held open with the little hooks, after which the parasite in most cases escapes without further manipulation. If the cysticercus is situated in the vitreous, the incision has to be carried through all the tunics of the eye, and a spoon may be necessary to remove the animal. The way of removing this parasite from other parts of the globe will suggest itself by the peculiar features of each case.

(*b*) *The foreign body is of iron or steel.* In this case the *magnet operation* is our chief reliance. It was successfully used by Dixon.[1] A piece of steel 1‴ in length was seen indistinctly in the anterior part of the vitreous four weeks after its entrance. It was drawn to the sclerotic with a large magnet, and extracted with an Assalini forceps through a scleral incision made with a lance-shaped knife.

William A. McKeown[2] tried in vain to get a chip of iron out of the vitreous with forceps. Then he introduced the tip of an eight-inch magnet into the vitreous towards the posterior pole. The foreign body was attracted by the magnet, but stripped off twice at the scleral opening; at the third trial it came out. This was followed by recovery. Since that time the magnet operation has been enthusiastically advocated. Instead of a permanent magnet used by McKeown, E. Gruening, and others, the electro-magnet soon came into general favor. A convenient pattern of a small electro-magnet, requiring only a quart bichromate element, constructed according to the design of J. Hirschberg,[3] of Berlin, is still most extensively used in its original form or with slight modifications,—for instance, by Simeon Snell,[4] of Sheffield. Many successful operations have been made and reported. The magnet has been successfully tried even when the foreign body could not be seen, and not only when the extrac-

[1] Dixon, Royal London Ophthalmic Hospital Reports, vol. i. p. 280.

[2] McKeown, British Medical Journal, June 20, 1874.

[3] J. Hirschberg, Centralblatt für praktische Augenheilkunde, 1879, S. 380, and very exhaustively in his papers "On the Extraction of Chips of Iron or Steel from the Eye," Archives of Ophthalmology, vol. x., 1881, pp. 369–398, and Ueber die Ergebnisse der Magnetoperation in der Augenheilkunde, Archiv für Ophthalmologie, 1890, Bd. xxxvi., 3, S. 37.

[4] Simeon Snell, British Medical Journal, May 28, 1881, and The Electro-Magnet, etc., London, 1883.

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tion was made soon, but four weeks or longer after the injury. The mode of application is through an incision in the ocular capsule, mostly meridional, between the external and inferior recti. The stripping off which occurred in the first case (McKeown's) is still an obstacle, to obviate which the drawing the lips of the wound apart, according to the method of Arlt in cysticercus operation (see above), is to be recommended, and can easily be done in magnet operations, if the hooks, as I have had them made and used, are of platinum and the handles of glass. These hooks are not influenced by the magnet, and can, by boiling or glowing, easily be kept aseptic.

The **indications** and **results** obtained with a small magnet are fully detailed by H. Hildebrand.[1] In general, it is advisable to try to remove the foreign body as soon as its presence can be ascertained. The diagnosis can be made with oblique illumination, with the ophthalmoscope by transmitted light, or by the indirect and direct methods, by a "sideroscope" (the best thus far is that of Asmus[2]), and by a large electro-magnet (Haab's is the best). Even if the foreign body cannot be seen, its presence can in most cases be ascertained by the sideroscope and the large magnet. A sudden pain is felt in that part of the eye towards which the foreign body is drawn by the magnet. In some cases the magnet will draw the chips of iron into or out of the opening through which they entered, or they are drawn from the vitreous into the anterior chamber.

If the foreign body has produced a good deal of inflammation, or is enveloped by blood, it is better to watch the patient and be guided by the reaction. If a small chip of iron has been adherent to the background of the eye for some time, it is advisable to leave it as long as it gives no trouble. Foreign bodies are known to have lain in the eye for many years without causing irritation, yet it is not infrequent that sooner or later they will work loose from their bed, sink to the bottom of the vitreous chamber or upon the ciliary body, and cause painful inflammation, perhaps sympathetic ophthalmia. Patients ought to be informed of this possibility, and urged to report at once when they feel anything wrong with the injured eye. When an iron particle has entered the eye, the sooner the magnet is applied the better are the chances of a successful removal. In some cases apparently hopeless a perfect result may be obtained when the turbidity of the media has subsided, after days, weeks, or even months during which the foreign body could not be seen. In one of Hildebrand's (*i.e.*, Dr. Mayweg's) patients,—Case No. IX.,—severe inflammation, intense pain, complete turbidity of the vitreous, and almost total loss of sight were present,

[1] H. Hildebrand, 66 Magnetoperationen mit erfolgreicher Extraction von 53 Eisensplittern aus dem Augeninnern, Archiv für Augenheilkunde, Bd. xxiii., 1891, S. 278, etc., and in abstract, Archives of Ophthalmology, vol. xxiii. p. 167.

[2] E. Asmus, Description of the Sideroscope, Archiv für Ophthalmologie, Bd. xl., 1894, 1, S. 280, and Archiv für Augenheilkunde, Bd. xxix. S. 126, Bd. xxxi. S. 49, and especially Bd. xxxi., Ergänzungsheft, 1895, S. 3.

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yet so excellent a result was obtained by the operation that on examining the patient seven years later Hildebrand could not distinguish which had been the affected eye. Air-bubbles in the vitreous are very rarely met with. They are easily recognizable, and a sure sign of the entrance of a foreign body into the eye.

Iron foreign bodies located in the anterior parts of the eye are oftener successfully extracted with the magnet than those located deeper.

Hildebrand has compiled 322 operations performed with the small magnet. In 80 patients the foreign body was located in the anterior parts of the eye. Of these, 13 ended unfavorably on account of purulent inflammation having set in before the operation; 67 operations yielded a good result.

In 74 cases the magnet was introduced into the interior of the eye, but the foreign body was not found.

In 174 cases the particle of iron was extracted.

23 of these (thirteen per cent.) led to phthisis bulbi.

26 (fifteen per cent.) necessitated subsequent enucleation.

Of 34 the results could not be ascertained.

The remainder, 91 cases, gave satisfactory results: in 29 cases preservation of the shape of the eye, in 62 cases more or less good vision.

Hirschberg (*Archiv für Augenheilkunde, l. c.*, S. 64) reports 13 successes among 100 operations of his own up to 1890, of which 4 were good, 3 moderate, and 6 slight results. He never saw sympathetic ophthalmia after his operations.

Comparing the results obtained by Mayweg and Hirschberg, we find: Hirschberg introduced the magnet into the vitreous in 65 cases, Mayweg in 51. Iron particles were extracted by Hirschberg in 29 cases (forty-four per cent.), by Mayweg in 38 (seventy-four per cent.). In 16 cases (thirty-one per cent.) Mayweg obtained useful vision, Hirschberg in 7 cases (ten per cent.).

Adolf Hürzeler[1] reports on 18 cases from the practice of Professor Haab in Zurich. The foreign body was in the posterior part of the eye,—*i.e.*, behind iris and lens. The small magnet was used up to 1892. In 5 cases (27.8 per cent.) the foreign body was not found; in 13 (72.2 per cent.) it was extracted. The wound was not sutured, "as experience has shown that under aseptic dressing it will close rapidly and well." The present writer can endorse this statement. The best vision obtained was 1/7 in one case; eight cases had very indifferent sight, movements of hand or counting fingers near by, and most of them had detachment of the retina; in two the shape of the eyeball was preserved; in seven (38 per cent.) the eyes had to be totally sacrificed. The experience of the present writer is not quite as bad. He has obtained permanently perfect sight in a few cases where the

[1] Beiträge zur Augenheilkunde, Die Anwendung des Electromagneten, Heft xiii., 1894, S. 20.

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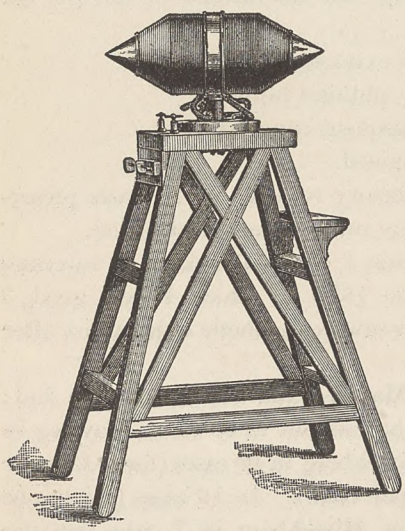
foreign body could be seen and extracted soon after its entrance.[1] The literature on this subject is copious.

A new phase in the development of the "magnet operation" has been introduced by Haab.[2] In consideration of the shortcomings of the small magnet, he constructed a large one, a Ruhmkorff coil, modified for the special purpose of extracting foreign bodies from the eye. The description, mode of application, and the results obtained up to 1894 are given in his paper published in Deutschmann's *Beiträge* (*l. c.*).[3]

The magnet rests on a wooden stand, a little over a metre high, and movable on casters (Fig. 51). It is held by a brass pivot fitting in a hollow cylinder standing in the centre of a solid brass platform, with which also the screws, wires, etc., are connected. A strong coil of insulated copper wire five centimetres in thickness surrounds a soft iron cylinder sixty centimetres in length and ten centimetres in diameter, ending in two conical tips. The coil weighs fifty-six kilogrammes and the iron core thirty kilogrammes. The magnet turns readily on its vertical pivot. The patient is placed on a chair which can be raised, lowered, and turned. His arms rest on a plank at the stand, so that his eye can be readily moved to and from the tip of the magnet. The instrument requires a constant current of from ten to twenty amperes and from sixty to one hundred and twenty volts. The current can be derived from any constant source of electricity, most conveniently from the street current in towns provided with electric lighting. The apparatus may remain connected with the power-source until the coils are no warmer than the hand. A bipolar interrupter and a safety arrangement are placed in the conduit before the magnet. The current must be interrupted in the coils, not by the stop-cocks on the side of the stand, as these serve to change the field-power. The apparatus is to be kept in a place protected from dust and moisture. Of late Dr. Haab has added to his instrument a current-reverser. A balancing board, turning on a horizontal axis in its middle, can be moved with the foot, closing and reversing the current by alternately pressing with the toes or the heel. Dr. Haab thinks that by reversing the current foreign bodies adhering to the tissue may become loosened.

FIG. 51.



Haab's large electro-magnet.

The mode of application of this powerful magnet is not simple. It

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[1] H. Knapp, Two Recent Magnet Operations,—One an Ideal Success, the Other a Total Failure, with Remarks. Transactions of the American Ophthalmological Society, vol. vii., 1894, p. 52.

[2] O. Haab, Bericht über die 22. Versammlung der ophthalmologischen Gesellschaft zu Heidelberg, 1892; *ibidem*, 1895, S. 191; Neuer Electromagnet, etc., Beiträge zur Augenheilkunde, Heft xiii., 1894, S. 68.

[3] At my request Dr. Haab has been kind enough to send me a detailed communication on the construction and application of the large magnet, as well as his experience with it up to the present time (October, 1897), allowing me to use it in the preparation of this article. I take this opportunity to thank him heartily for his kindness.

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has to be learned, and some of the difficulties in its handling have been pointed out by recent publications. The main advantage the large magnet has over the small one is that its power lends it a considerable action at a distance, whereas the small magnet is essentially a magnetic probe, which does excellent service when its tip touches the foreign body. In its way, it frequently supplements the large magnet most happily. The latter draws the chip of iron from the depth of the eye into the anterior parts, behind the iris or into the anterior chamber, where the tip of the small magnet can reach and easily extract it. The large magnet, if its power-lines are properly directed, will draw the foreign body from the background either through its passage of entrance or on a new way to the surface, where it is accessible. This will be done also when the foreign body is invisible.

Should the presence of an iron or steel foreign body in the interior of the eye be doubtful, the large magnet is the most practical instrument to decide the question one way or the other. In most cases when the current is closed the foreign body flies to the wall of the eyeball with a sudden impulse, causing more or less pain. If this is the case the presence of iron in the eye is demonstrated, but the negative result of the experiment is not conclusive: the splinter may be so firmly embedded in the tissue that the force of the magnet does not move it. The greater this force the less deficient will be its action. Haab says, "He that has the strongest magnet will have the best result." When the magnet fails to demonstrate the presence of a foreign body, the sideroscope (of Asmus) is in order. This instrument is a marvel of delicacy, but to handle it requires the patience of a saint.

Haab dwells on the advantage of the large magnet in drawing out foreign bodies from the depth of the eye without churning the vitreous. This is truly an advantage, for the repeated introductions of the electrode of the small magnet, and the blind search for a needle in a hay-stack, injure the vitreous, so that all the pleasure and applause derived from the successful extraction of the splinter are more than counterbalanced by the subsequent development of detachment of the retina and shrinkage of the eyeball.

Before the operation is begun, the surgeon and all by-standers must put their watches in another room, lest the great force of the magnet, when the current is turned on, magnetize certain parts and spoil the watches. We must know exactly in which direction and with what strength we shall let the magnetic force act on the eye. Frequently it will be in the direction of the track of entrance, especially if this be at the periphery of the cornea or on the sclerotic. The head is steadied by the hands of the operator, and the eye very gradually approached to the pole of the magnet. If there is no sudden sensation of pain, indicating the impact of the splinter against the ocular wall, we should not at once give up the experiment, but let the current continue, reversing it now and then, and move the eye slowly in different directions, for by these manœuvres foreign bodies have been seen to become loose, and in about ten minutes follow the traction of the magnet.

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If the foreign body has entered the globe at the corneo-scleral border without injuring the lens,—the most favorable case,—we may advantageously enlarge the wound if it be small, which means that the foreign body is also small; we slowly approximate the eye to the pole of the magnet and apply the tip directly to the wound, for the smaller the foreign body the less violently it will be attracted by the magnet. The foreign body then is very apt to appear in the wound attached to the tip of the magnet. Eighteen months ago this occurred in a boy who came to me in the just-described condition. Thinking his a fit case for the Haab magnet, and having at the time no strong magnet in my possession, I took him to the New York Eye and Ear Infirmary, and asked Dr. J. E. Weeks, my former assistant, to try the magnet. The foreign body came out at the first attempt, smoothly and without any clash. The case proved a complete success.[1]

In directing the current to this region, especially if the foreign bodies are comparatively large, we must be very cautious and approach the patient only very slowly to the magnet, for the foreign body may be attracted with such force that it will burrow in the ciliary body, from which it may not be possible to extricate it, and if we enlarge the wound and apply the current again, the ciliary processes are liable to be pulled out with the foreign body, bruised and lacerated. The same, in a less degree, will occur if we draw the foreign body forcibly into the root of the iris.

If there is no track left from the entrance of the foreign body, we should direct the pole of the magnet to the centre of the cornea and move the eye very gradually towards the magnet. The body will then in many cases be drawn from the depth of the eye to the posterior pole of the lens, and, if the lens be unbroken, move around its posterior surface, perforate Zinn's zonula, and enter the posterior chamber, where it will make its presence known by a circumscribed bulge. We should try to draw it through the dilated pupil into the anterior chamber, holding the eye in such a way towards the foreign body that the traction lines of the magnet pass obliquely through the anterior chamber in the direction of the foreign body. Should we not succeed in this attempt, we would have to incise the cornea in front of the foreign body, make the iris prolapse, remove the foreign body with the large or small magnet, and reduce the iris. If we do not succeed, we would have to extract the foreign body together with the iris, or after an iridectomy has been made.

Authors seem to be averse to making an incision into the sclerotic even when the foreign body is known to be in the vitreous. They seem to prefer drawing it into the anterior chamber and then remove it with a small magnet. I should think a small opening between the inferior and external recti, the place of election with the small magnet, would answer also to the use of the large, especially if we separate the lips of the wound with delicate platinum wire hooks.

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[1] John E. Weeks, The Removal of a Piece of Steel from the Interior of the Eye by the Electro-Magnet of Haab, Archives of Ophthalmology, 1897, vol. xxvi. p. 85.

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Haab, in his written communication to me, mentions a case where a steel splinter was firmly adherent to the retina upward and inward from the optic disk and did not yield to the attraction of the magnet. Under the guidance of the ophthalmoscope, he thrust a discission-needle into the eye, cut the splinter loose, drew it behind the iris, and extracted it through a small corneal incision.

The removal of foreign bodies from the anterior parts of the eye—cornea, iris, anterior chamber, and lens—is easy, and requires no special description after the foregoing remarks.

Haab writes me that he has used the magnet, thus far, in about eighty cases. On forty-three he reported at the Heidelberg Congress in 1895. The further cases yielded the same results. He continues to be satisfied with the results of the large magnet operations, and has used the small magnet only rarely, yet thinks it would be well always to have it at hand.

The sideroscope and the large electro-magnet render a group of cases amenable to treatment which without them would be rather unpromising. If the bulk and the cost of the instruments will prevent their popularity, they should at least make a part of the armamentarium of all larger ophthalmic hospitals.

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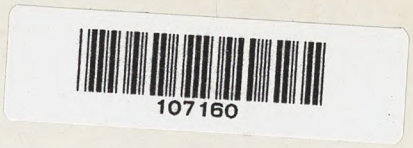
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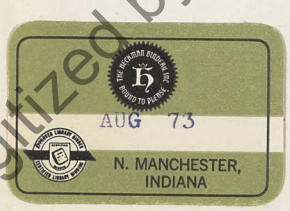
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